THE MEDICAL JOURNAL OF AUSTRALIA

VOL. II.—12TH YEAR. SYDNEY: SATURDAY, NOVEMBER 7, 1925. No. 19.

Table of Contents

X RAYS AS AN AID IN THE DIAGNOSIS OF THE PATHOLOGICAL GALL BLADDER.[1]

By A. SYME JOHNSON, M.B., B.S. (Melbourne.),
Honorary Assistant Radiologist, Perth Hospital.

THE right epigastrium may be well termed the region of the surgically vague. It is tenanted by several of the most important digestive and metabolic organs.

Some of its pathology has very definite and clear cut clinical manifestations, but it so happens that many of its abnormal states are peculiarly difficult to diagnose and of the latter probably the most vague and difficult of all are those which have their origin in the gall bladder.

In our surgical experience we learn at an early stage the relative importance of signs as compared with symptoms and quite a proportion of our patients present themselves for examination with a story of symptoms referable to this region and often the examination fails to disclose a single sign. Moreover the symptoms themselves may be located by the patient in regions which are anatomically at a considerable distance from the area we are discussing. It is in consequence of this that so much difficulty is found in reaching definite and adequate diagnoses and that we must explore every avenue of investigation which may lead us nearer the truth.

One such avenue is found in the examination by means of X rays and before proceeding to some discussion of the signs which may be brought to light during a comprehensive examination by this means, I would like to touch briefly on the past history of the application of the Röntgen examination to pathological states of the gall bladder.

This term "pathological states of the gall bladder" has been chosen advisedly for reasons which will appear.

It is only about twenty-five years since gall stones were first disclosed on a skiagram and it is interesting to note that in at least one of the earliest cases the shadows shown on the plate were not recognized for what they really were, inasmuch as the radiologist did not conceive that gall stones might be shown by the rays. I think I am correct in saying that amongst the earliest cases was one reported by Thurston Holland about 1905 in which he found his shadows on the plate, but did not interpret them as gall stones. The patient was submitted to

[1] Read at a meeting of the Western Australian Branch of the British Medical Association on August 19, 1925.

operation during which two gall stones were found and removed. On comparing these with the skiagram it was seen that they definitely corresponded with the shadows thereon.

A few isolated instances appear in the early literature and were regarded more or less as curiosities. The next stage is one in which more and more such cases are reported. It now became the aim of the radiologist to demonstrate the calculi themselves on the plate.

This was a great pity, for the few successful demonstrations were accompanied by numerous failures and "negative" reports were shown at operation frequently to be entirely misleading. Calculi were found by the operator in many instances in which they had been pronounced absent by the radiologist. The result was naturally to cast much discredit on the method and many prominent surgeons gave it as their opinion that the method was misleading and therefore dangerous. This point of view has to some extent persisted to the present time and I repeat that this search for calculi was very unfortunate, for the fault lay not in the method itself, but in the fact that the radiologists were searching only for calculi and in the state of technique and apparatus then existing such a procedure was predoomed to failure.

Bearing this initial check in mind, let us continue our brief historical review, for in so doing we shall lead to a logical conclusion as to the present day value of the method.

The next significant period is about the year 1914. By this time the opaque meal had become fairly established and was being used by the progressives as a means of elucidating the various dyspepsias.

In this it became a method of value, but its application soon proved to be much wider, for many signs came to light which made no apparent connexion with the stomach or intestinal tube as such, but which were seemingly the result of nervous and mechanical influences arising from other abdominal organs. As always in the history of human progress data accumulating in the records of hundreds of investigators began to be grouped into orderly sequence, their significance was at first assumed, then proved and finally accepted facts of known causation became the common property of us all.

However this seems to need some correlation with our subject, so in the words of Kipling "let a plain statement suffice." The opaque meal as used originally for the investigation of gastric phenomena has afforded us a number of signs definitely related to pathological states of the gall bladder.

It is now the firm opinion of many of the world's leading radiologists and equally of leading surgeons that these so called indirect signs of gall bladder mischief as disclosed by the opaque meal are of greater importance than the direct evidence of calculi or of the gall bladder demonstrated on the film itself. Personally I strongly believe this dictum to be true.

Direct Evidence of Gall Bladder Disease.

The direct signs of the pathological gall bladder itself are only two in number: (i.) Visible gall stones and (ii.) a visible pathological gall bladder. For the first if undoubted shadows of calculi appear on the plate, the evidence is summary and no further corroboration is necessary. Unfortunately even today with our more perfect technique and apparatus it is frequently very difficult and often impossible to secure films on which gall stones can be identified, even when they are present in the body of the patient.

To the second namely, the visible gall bladder the same qualifications apply. With care and attention to all the details of preparation of the patient and of the actual radiological technique, one can demonstrate the bladder in outline in a fair proportion of cases. Many patients, most in fact are of the sthenic class and not a few are actually obese and their bulk adds much to the difficulty of securing good skiagrams, even when the greatest care is taken.

From this it will be understood that the detection of shadows is of much value; yet the absence of shadows is of little value at all from a diagnostic point of view owing to the known difficulty of producing them. In fact were we to limit our search to these two points, as was done in the past to the first of them, we should have to acknowledge that only in about 30% of cases could we give assistance that was worth while to the clinician.

Fortunately for everyone concerned another and larger and as I believe more important group of data is within our reach.

Indirect Evidence of Gall Bladder Disease.

Reference has been made to the so called indirect evidence and to the fact that it can be elicited in the course of a barium meal examination. The evidence naturally is found in those organs occupying the right epigastrium and is best described in the order in which it is encountered during such an examination. It is reasonably subdivided into three groups of signs: (i.) Those affecting the stomach; (ii.) those affecting the duodenum; (iii.) those affecting the colon.

Signs Affecting the Stomach.

Shape and Lie of Stomach.—The patients being frequently of the robust sthenic habit the stomach is often found to be of the oblique lying, steerhorn type rather than the normal fish hook shape. This brings it nearer the gall bladder area and in closer relation to the sphere of activity.

Spasm of Stomach.—Frequently one meets with a certain type of spasm which takes the form of a cylindrical narrowing of the distal end of the stomach closely resembling the shape and dimensions of a German sausage. Its origin is undoubtedly reflex, for it is frequently absent in the first few minutes after ingestion and in my experience usually sets in after the pyloric canal opens. It is fairly constantly associated with the pathological gall bladder.

The Semilunar Impression.—The next sign of consequence takes the form of a semilunar impression on the outline of the stomach usually on the antrum. This is a common type of the indirect

evidence and when found is strongly suggestive of a pathological gall bladder.

It is frequently demonstrated only in one position of the patient, but should always be looked for, as when found it is of much value. It takes the form of a smooth rounded incurve of the stomach or duodenal wall and without doubt corresponds exactly to the curve of the gall bladder itself. The possible fallacies are few in number and occur but rarely.

Signs Affecting the Duodenum.

The Semilunar Impression.—Of the type of evidence affecting the duodenum the commonest and perhaps most valuable is that just described for the stomach, namely the semilunar impression. It is much more commonly found in the duodenum than in the stomach owing to the closer approach of the former to the gall bladder. It is further more frequently found affecting the first part or cap and much more rarely the second or descending part of the duodenum.

Fixation of the Duodenum.—Normally the first part of the duodenum has a small but definite degree of mobility. Often in cases of suspected gall bladder mischief it is found to be anchored close under the right costal margin, the fixation being due to adhesions from a pericholecystitis. The second part may be drawn over to the right or its vertical line of descent angulated to the right.

Distortion of the Cap.—Distortion of the cap also has its origin in adhesions and frequently takes the form of a series of capes and bays in the outline of the cap due to interference with the barium filling by dragging or pressure of the adhesions.

Changes in the Ampulla of Vater.—Normally no barium ever enters the mouth of the duct, but sometimes the latter is dilated and receives quite a perceptible amount of barium which it tends to retain. While not common, this sign has proved to be very strong evidence of gall bladder or pancreatic mischief. The cause of the dilatation is not clearly known, but may be due to the previous passage of a stone or to retraction by adhesions.

Fixation of Jejunum.—In a few cases loops of the jejunum, which normally belong rather to the left epigastrium, are found more or less fixed over to the right near the gall bladder site.

Signs Affecting the Colon.

Position of Hepatic Flexure.—If the hepatic flexure is high and fixed, particularly if the ascending colon and caecum are of normal length yet suspended at an unusually high level, it is suggestive of inflammation near or at the gall bladder site.

"Gas Trap."—Often a small "picked up" portion of the hepatic flexure is seen, the apex of the deformity having the form of a dunce's cap and usually retaining gas even when the rest of the lumen of the gut is normally filled with barium emulsion.

The Hepatic Pseudoflexure.—The hepatic pseudoflexure is quite frequently found. The name is derived from the fact that part of the transverse colon usually from five to 12.5 centimetres distal to the true hepatic flexure is looped up by an adhesion to the neighbourhood of the gall bladder and gives the impression of a double hepatic flexure.

The Value of Indirect Evidence.

I have now enumerated some eleven types of indirect evidence. It is probable that not one of them is of paramount importance or in any way pathognomonic of the gall bladder mischief. Their great value lies in their number, the mutual support they give to one another and the ease with which they are demonstrated and recorded. If only one or two of the important pieces of evidence are present, the diagnosis is suggested; given a sufficient number present in any one patient, the conclusion is almost absolute. It must be noted that they do not and cannot distinguish between the presence or absence of stones, but when all is said and done this distinction is not usually of the first surgical importance. In all probability a gall stone is only an incident in a cholecystitis.

These X ray signs have no present known relation to the clinical symptoms or to their intensity. It is here that I would like to emphasize what appears to be the great value of the X ray examination. The cases clinically may be vague in the extreme, yet the X ray signs may be present in such character and number as to bring us to an absolutely definite diagnosis. The converse is without doubt also true, but in such cases the help of X rays is needless. In the border line or "half and half" case if one may so term it, the radiological examination (if taken in conjunction with the clinical signs) will probably elicit sufficient data to justify the diagnosis and the treatment, though if taken by itself, it too may be indecisive.

It is interesting to obtain some idea of the actual results to be expected from the method. The following statistics were not selected, but merely the first to come to hand. They are the work of four prominent men and represent two published tables averaged together by myself. Every patient in the series was operated on and the X ray findings confirmed or contradicted with the results shown below.

Total cases reported	209
Errors	22
Correct	187
Percentage of error	10.7
Percentage correct	89.3

From these figures it will be appreciated that the method is a very real help and may be relied on to give important assistance in nine cases out of ten.

Cholecystography.

It is pleasing to relate that the end is not yet in sight, for within the last two years a fresh method of investigation has been introduced by Graham. This process makes use of the fact that certain salts of tetrabromphenolphthalein are filtered from the blood stream by the gall bladder.

About 4.5 grammes of a soluble salt, usually the sodium salt of this compound is injected intravenously and within three to five hours the bromine content of the compound has been collected in

sufficient concentration by the gall bladder to allow of its outline being projected on to a film in much the same way that we photograph the barium filled stomach. This is possible on account of the radio-opacity of the element bromine.

The greatest opacity of the gall bladder is usually found at sixteen to twenty-four hours.

The method is at present applicable only to hospital patients, for certain undesirable symptoms may arise. While it is full of promise it has not yet reached the stage of being applicable as a routine measure. Probably it will be somewhat modified and even should it never become a routine it holds out hope of achieving for the gall bladder the same wonderful opportunity of investigation, both anatomical and physiological as well as pathological, as has been secured by the opaque meal method for the stomach and intestine.

Its utility will be much increased if some modification of the method can be made which will permit of oral administration, possibly by the use of some less toxic drug. Indeed already early reports of such a method have been received.

Conclusion.

In conclusion, I should like to appeal to you all for cooperation as between the physician and surgeon on the one hand and the radiologist on the other. So great a man as William Mayo has said that X ray methods of examination are only in the daguerreotype stage as yet. If that is so, there is yet an enormous field of utility before us. It is only by mutual help and exchange of ideas that we shall advance rapidly towards our common ideal. It is of the greatest help to the X ray man when he is informed of his mistakes, for he may then take warning and possibly may find where he is wrong. He cannot personally follow every patient to operation and so learn the confirmation or contradiction of his conclusions. He therefore very much appreciates a brief account of the surgical findings or of the medical progress of the patients that have been sent to him.

THE PHYSIOLOGY AND SIGNS AND SYMPTOMS OF THE TOXÆMIAS OF PREGNANCY.[1]

By J. Leon Jona, D.Sc. (Adel.), M.D. (Melb.), M.S. (Adel.),
Melbourne.

I first want to thank the Melbourne Permanent Committee for Post-graduate Study for the honour they have paid me in asking me to deliver this lecture.

The term "toxæmias of pregnancy" embraces a number of conditions, some of which are well defined, which are supposed to be due to a toxin or toxins circulating in the blood of a pregnant or parturient woman, production of which is in some way connected with pregnancy or the products of conception. The condition which will be dealt with in the present discourse will be salivation, vomiting of pregnancy, nephritis of pregnancy, acute yellow atrophy of the liver, eclampsia and a number of conditions which Whitridge Williams[1] includes under the heading of "presumable toxæmias."

[1] Being a lecture delivered during the obstetrical course of the Melbourne Permanent Committee for Post-graduate Study on June 9, 1925.

Salivation.

Salivation in its mild forms is not uncommon, but in extreme forms may be very serious, causing great loss in weight and, as a result of the physical inconvenience caused by the ptyalism, having a profound effect on the nervous system. It may recur in subsequent pregnancies. The pathology of the condition may be nervous, either reflex (though I confess to a personal difficulty in seeing how such a reflex disturbance would be set up) or local, similar to the "paralytic" secretion produced on section of the nerves to the salivary glands.[2] On the other hand it may be toxic. By the injection into the dog of toxic extracts of decomposed liver, placenta and spleen I have been able to set up a profuse secretion of saliva with small doses and an inhibition of salivary secretion with larger doses, which even inhibited the sialagogic effect of pilocarpine[3] (see Graphs I. and II.). Similar effects were produced at the same time on the secretion of tears, sweat and the activities of the glands of the mouth and also the pancreas. Atropine is advised as a treatment and it must have a beneficial effect where the pathogenesis of the condition is nervous, but if the toxin acts directly on the cells of the salivary glands, then it is obvious that atropine is valueless; this is found to be the case in some patients. In these cases the treatment is to eliminate the toxin by all the eliminative measures as are at our disposal, since we cannot neutralize the toxin because we do not know what it is or where or how it is produced.

Vomiting of Pregnancy.

The early vomiting of pregnancy occurs in one-third to one-half of all pregnant women[4] and is generally not serious. It occurs usually from the sixth to the twelfth week of pregnancy and as a rule disappears spontaneously after the third month. While it lasts it only manifests itself as a more or less transient morning sickness which can be cured by regulation of diet and mode of living, small frequent meals and the eating of a hard dry biscuit immediately on waking.

There are three accepted types.

The Neurotic Type.

The neurotic type is similar to the condition dealt with above and regarded as due to disturbance of endocrine activity especially *corpus luteum* and the treatment is psychotherapy.

The Reflex Type.

The reflex type is due to some genital trouble or to a retroverted uterus especially if there is a tendency to incarceration. This is a condition which must be carefully considered as, if neglected, it may lead to abortion.

I remember being asked to see a patient who had been suffering from persistent vomiting. She had had her appendix removed and still vomited; then her gall bladder was first drained and then removed and still she vomited. I examined her and found a large retroverted uterus well down below the promontory of the sacrum. I replaced the uterus with the patient in the knee-chest position, introduced a ring pessary and the vomiting ceased.

The Toxæmic Type.

The toxæmic type, although denied by Comyns Berkeley,[5] undoubtedly exists as a definite entity. Williams[6] states that it occurs in one in one thousand pregnancies. The symptoms are persistent vomiting in spite of all medical measures and general malaise, the patient is usually depressed on account of the persistence of the vomiting and the patient loses weight and strength. The urine has a high ammonia content which in 4% to 5% of the patients may reach 20-50%.

Sometimes and especially in fatal cases it is associated with liver lesions and degenerative kidney lesions. I regard this condition as due in part to a failure of the liver cells to convert ammonia into urea. Soda solutions injected into the rectum and in one case even sodium bicarbonate solution injected into the veins had no effect on the ammonia coefficient or on the reaction of the urine.[7] In one case although the patient starved some days before and after abortion due to pernicious vomiting, the ammonia coefficient fell from 33% to practically normal, even while the patient was starving. As a rule there is acetone in the urine, but this, of course, may be due to the starvation or to the toxin. In the extreme forms, however, the vomitus may contain coffee grounds or bright blood due to a gastrostaxis.

It is an established fact that toxins have an affinity for calcium[8] and so it is quite possible that this vomiting of blood is due to an extreme toxæmia in which the gastric and intestinal mucosa has been deprived of calcium, its vitality thus impaired and the mucosa and its blood vessels damaged by the free hydrochloric acid in the gastric juice. The condition can be imitated by the administration to a dog (under anæsthesia) of some neutral potassium oxalate.

The treatment adopted in the early stages is starvation, lavage of the bowel and general eliminative treatment.[9] When, however, the patient is beginning to vomit blood, her position becomes serious and I believe that one should immediately induce abortion.

Acute Yellow Atrophy.

Acute yellow atrophy may occur as early as the sixth week, but generally occurs at the end of pregnancy or in the puerperium. The disease is characterized by a rapid diminution in the size and weight of the liver which in quite a short time may be half its normal size with wrinkling of the capsule. The liver tissue is soft and may be red or yellow in colour and each lobule has a red centre of necrosis. The kidneys show the signs of an acute nephritis, the tubules being degenerated, but not the glomeruli. The symptoms may be acute abdominal pains, intense headache and sometimes vomiting and purging. The patient becomes jaundiced as a rule and may exhibit a sudden rise in temperature with general malaise, an erythematous or purpuric rash or even hæmorrhages from the nose, kidney, stomach or any mucous surface may appear and the patient becomes torpid, delirious, comatose and may even have convulsions. The vomitus is frequently blood-stained and sometimes contains coffee grounds. The urine is diminished in amount and contains albumin, casts, blood and often leucin and tyrosin which may float on the surface as a skin and are recognizable under the microscope. The total nitrogen content may or may not be diminished. The ammonia coefficient is greatly raised. The condition is a very serious one and is due to necrosis of the liver cells commencing at the centre of the lobule. The signs and symptoms in a way overlap those of eclampsia, such as unconsciousness and convulsions and the presence of acetone in the urine, but the presence of bile is regarded as pathognomonic of the disease. The symptoms and signs are apparently due to a failure in function of the liver cells, hence the high ammonia content of the urine as well as the accumulation of bile in the blood due to the failure of the liver cells to excrete it. I believe that the best treatment is immediate delivery by forceps if the *os uteri* is fully dilated[10] or by Cæsarean section if the *os* is not dilated or dilatable. I have performed a Cæsarean section on a patient with this condition and the result was eminently satisfactory, particularly when regarded from the mother's standpoint. When anæsthesia is induced for these patients, chloroform should never be used, as it in itself can produce an effect on the liver similar to acute yellow atrophy.

Nephritic Toxæmia.

Nephritic toxæmia generally occurs in women who have had chronic nephritis before becoming pregnant and must be distinguished from the albuminuria of preeclampsia. It generally comes on in the later stages of pregnancy and is characterized by lassitude, general malaise, headache, drowsiness, vomiting, general œdema and occasionally the eye symptoms of albuminuric retinitis, as well as cardio-vascular changes, such as hypertrophy of the left side of the heart and arteriosclerosis and a high blood pressure. On the other hand, however, there may be no symptoms or signs whatever and the only abnormality noted is the albuminuria, whereas a third type may be more truly a toxæmia with toxic spoiling of the cells of the kidney, such as is seen in phosphorus poisoning with pale yellow kidneys with more or less microscopical effusion of blood under the capsule. It is generally held that the condition developes in those patients whose kidneys, although diseased, are able to functionate more or less satisfactorily under ordinary conditions, but give way under the additional strain imposed on them by the condition

of pregnancy when the mechanical effect of the large uterus of late pregnancy as well as the necessity for the excretion of fœtal and increased maternal waste products tell on the damaged kidney. In the chronic forms red and white infarcts occur in the placenta, hence the nourishment of the child is impaired and it often dies or is born prematurely. The urine is normal in amount or may even be increased and contains large amounts of albumin as well as casts, which may persist for months after labour is terminated. The total nitrogen, urea and ammonia coefficients are generally unchanged. If the patient is passing large quantities of urine and the general symptoms are not getting worse, I consider that the pregnancy should be allowed to go on in spite of the large amount of albumin present.

Eclampsia.

Definition.

According to the Bertillon system[11] of nomenclature (1909) "puerperal albuminuria and eclampsia" include nephritis of pregnancy, puerperal nephritis, puerperal uræmia, eclampsia in women in labour, epileptoid convulsions of women in labour, puerperal tetanus, puerperal coma. In 1909 the following were added—puerperal nephritis, puerperal coma.

Such an unwieldy definition is difficult to apply to practical purposes. The Rotunda definition[12] postulates convulsions in pregnancy, labour or puerperium characterized by unconsciousness and generally accompanied by albuminuria, while Whitridge Williams[13] states that the only condition pathognomonic of eclampsia is a degeneration of the cells of the liver commencing with those cells on the boundary of the lobule, whereas in acute yellow atrophy the degeneration of the cells commences at the centre of the lobule, so that the absolute diagnosis can only be made *post mortem*. The condition of eclampsia is generally ushered in by a stage of preeclampsia, although occasionally patients are seen in whom the eclamptic condition developes quite regularly without any preeclamptic or premonitory symptoms. The condition of preeclamptic toxæmia may extend over a period of hours or days and is characterized by a more or less severe headache, lassitude and more or less general œdema. The patient may also have violent epigastric pain and visual disturbances which may vary from slightly impaired vision to absolute blindness. Albuminuric retinitis is sometimes present. The urine is often diminished in amount and may contain albumin or even blood. The patient may have hallucinations and exhibit symptoms of insanity or may pass from a condition of drowsiness into coma. The blood pressure sometimes rises to two hundred millimetres of mercury or more. Eclampsia may supervene even when urinary symptoms are improving.

The incidence of eclampsia varies in different countries and in the different cities of one country. Whitridge Williams[14] gives the average rate of about one among one hundred patients in hospital or about one among six hundred patients who were in hospital at the onset. Some years ago I analysed the eclampsia statistics in various cities and as this was before the introduction of regular antenatal treatment, the figures are comparable. Melbourne Women's Hospital had an incidence rate of 1.84% for 1913-1915, whereas the New Zealand Government maternity hospitals in 1914 had an incidence rate of 0.24%,[15] while in New South Wales subsidized hospitals in the metropolis had an incidence rate for the seven years 1908-1914 of 4.1%.[16] The incidence rate at the Women's Hospital in Melbourne for the years 1921-1924 was 1.57%, at the Royal Hospital for Women, Sydney, it was 1.3% and at the Crown Street Women's Hospital, Sydney, it was 0.76%. The rate, however, varies at different times. Cassamajor[17] in Tarnier's clinic in Paris found an incidence of one in forty-seven in 1872, of one in seven hundred and thirty in 1882 and of one in one hundred and thirty in 1891.

There is also apparently a seasonal variation as pointed out by Dr. R. H. Morrison[18] some years ago, who found that of two hundred and forty-five cases analysed one hundred and thirty-one occurred in the five cold months from May to September, giving a mean of twenty-six per month; one hundred and fourteen occurred in the seven warm months from October to April, giving a mean of sixteen per month. It is said that it may occur in the third month[19] but it generally occurs after the sixth month. From 70% to 80% occur in *primiparæ*,[20] from 5% to 8% occur in twin pregnancy, whereas the incidence of twins is 1.5%. During the years 1913-1915 in the Women's Hospital, Melbourne, the frequency in *primiparæ* was eclampsia in 54.7% and twins in 3% of the patients. Hydramnios accounted for most of the rest. Sometimes, as previously mentioned, there may be no preeclampsia, the condition developing without warning. Generally the actual convulsions are preceded by a stage of premonitory symptoms in which the patient may be unreasonable, irritable, restless and have irregular twitchings as a preliminary to the onset of the convulsions, preceding which there is a fixed expression in the eyes which soon begin to roll from side to side. The lids are rapidly opened and closed, the pupils are generally dilated, the *alæ nasi* twitch and the mouth twitches and is convulsed; the patient chews her tongue and then has twitchings of the face, arms, body and legs. The convulsions at first are tonic and then clonic; the patient gradually passes into a condition of coma. The progress of this march can be seen on reference to the cerebral localization of these various functions, the toxin apparently causing irritation of the cortical centres.[21] The convulsions may last a few seconds to two minutes. The patient's breathing is stertorous, the face congested and flushed, the patient foams at the mouth and often bites her tongue. The clonic stage passes into the third stage of coma in which death may supervene. The patient may have only one convulsion and the condition pass off and yet she may have two hundred convulsions and recover.[22] According to Lichtenstern a patient may have eclampsia at the seventh month, recover

from it and then go on to full term and have a normal confinement.[23] The patient's general appearance is also more or less characteristic. There may be œdema of the skin of an indurating type producing thickening of the integuments. The face looks "bloated" and is swollen and expressionless. There may also be vomiting.

Gibbon Fitzgibbon[24] says that preeclampsia seldom developes into eclampsia if within six hours of the starting of treatment eclampsia has not developed.

During a fit the blood pressure may reach 240 to 260 millimetres of mercury and the temperature may rise even to 42.4° C. (109° F.), but in case of recovery the temperature falls to normal within twenty-four hours. The cause of the rise in temperature has been attributed to either the toxin of eclampsia stimulating the thermal centres or to infection. I am inclined to believe that the rise in temperature is due either to increased heat production due to the convulsions or to the liver cells breaking down and liberating amino-acids which have a sympathomimetic action and thus cause vaso-constriction of the vessels of the skin with diminution of heat loss and consequent rise in temperature. A vaso-constriction of the skin can and does produce rise in body temperature.[25] I have seen cases of high temperature resulting from increased intraabdominal tension and consequent impairment of liver function. The temperature came down when the tension was relieved. After a fit the patient is unconscious and has no memory of the fit or of events immediately before or after the fit. In addition to dimness of vision, flashes before the eyes and blindness, the patient may be affected by hemianopsia.[26]

The convulsions may come on before labour sets in, during labour or after labour has terminated. The statistics for these classes are given by Whitridge Williams[27] as *ante partum* 55%, *intra partum* 22% and *post partum* 23%.

In my own experience there is a smaller proportion of *post partum* eclampsia.

Pathology of Eclampsia.

Regarding the pathology of the condition the urine as a rule is definitely diminished and generally contains albumin and casts; blood and hæmoglobin may be present. Acetone is usually present and is due to impairment of the fat metabolism, although I had one patient at the Women's Hospital, Melbourne, suffering from preeclampsia and although the urine was becoming normal, the albuminuria diminishing and acetone had disappeared, she developed eclampsia. The deprivation of food generally in the preeclamptic condition as well as the taking of carbohydrate foodstuffs which are often the main foods of the toxic patient, also induce acetonuria.[28]

As regards the nitrogen of the urine the total nitrogen is reduced, but in addition the amount of nitrogen excreted as urea is often less than half of the normal and the ammonia coefficient rises. There is also an increase in amino-acids and purin bases.

The Ureters.

The ureters are often dilated. A common (and according to Williams[27] constant) condition found is hæmorrhagic hepatitis in relation to the smaller portal vessels, as well as hæmatomata just under the capsule of the liver.

The Brain.

In the brain signs of œdema, hyperæmia, anæmia, thrombosis, apoplexy or thrombi in the smaller cerebral vessels are seen. According to Levant and Portes[29] hæmorrhage in the nerve centres occurred in forty-one patients among one hundred and eighty-three suffering from eclampsia out of a total of 55,488 obstetrical patients.

It occurred in the meninges in twenty patients, in the cerebro-spinal fluid in ten, in the cerebral tissue in eleven, in the pedunculi in three, in the bulbo-protuberal region in one and as a diffuse hæmorrhage in one.

The Heart.

A degenerative myocarditis is encountered, while œdema of the lungs, giant cells from the placenta in the capillaries and later broncho-pneumonia are common.

In fatal cases the commonest causes of death are heart failure, œdema of the lungs, broncho-pneumonia or sepsis. Eclamptics are particularly prone to sepsis and it has been noticed that after Cæsarean section for eclampsia the skin wound not infrequently breaks down in whole or in part.

In patients who recover, a very noticeable sign is diuresis; the patient often passes enormous quantities of urine, apparently getting rid of the water which appears to be stored in the tissues during the eclampsia. This condition of water storage in the tissues rather reminds me of what has been found by Dale[30] and others to occur in surgical shock. Consciousness generally returns fairly soon, but the blindness may persist for days and the impairment of vision for a considerable time, although the eye fundi appear quite normal. The albumin disappears in a few days after eclampsia, whereas in cases of nephritis toxæmia it persists more or less permanently.

Ætiology of Eclampsia.

As regards the ætiology of the condition numerous theories have been enunciated of which the following have received the greatest attention:[31]

(i.) That it is a disorder of the nervous system.

(ii.) That it is due to uræmia (Lever, 1843).

(iii.) That it is due to ammonium carbamate in the blood (Spiegelberg, 1870).

(iv.) The Traube-Rosenstein theory that it is due to anæmia and œdema of the brain.

(v.) The theory of Delore and Rodet (1884) of bacterial invasion; more recently J. E. Talbot[32] has enunciated a similar theory and considers that the placental infarct is the result of hæmatogenous infection of the placenta.

(vi.) Rivière evolved the autointoxication theory, the heaping up of toxins in the blood. This conception can be subdivided into seven groups:

(*a*) That it is due to products of fœtal metabolism. F. von Bemuth and F. Goebel[33] found that in the starving newborn babe there was an abnormally high percentage of amino-nitrogen in the blood and urine.

(*b*) That it is caused by the entrance of fœtal or placental elements into the maternal circulation, producing syncytiotoxin which should normally be neutralized by maternal syncytiolysin.

(*c*) That it is a manifestation of poisoning by substances formed or retained in the placenta.

(*d*) That it represents a disturbance of maternal metabolism, including intestinal intoxication. Normally, according to Mahnert,[34] in pregnancy there is a decreased oxidation of fats, carbohydrates and proteins. The respiratory quotient never reaches normal and there is decreased elimination of carbon dioxide especially on a protein diet. These changes in oxidation lead to a tendency to acidosis.

(*e*) That it is a form of anaphylaxis.

(*f*) That it is a mammary toxæmia (Sellheim). This theory is based on the parturient paresis or milk fever of cows which is relieved by injecting oxygen or air into the udders. This condition is now generally regarded as due to sepsis and the results due to maintenance of drainage.

(*g*) That it is due to thyreoid insufficiency, parathyreoid insufficiency, excess sarcolactic acid are other theories.

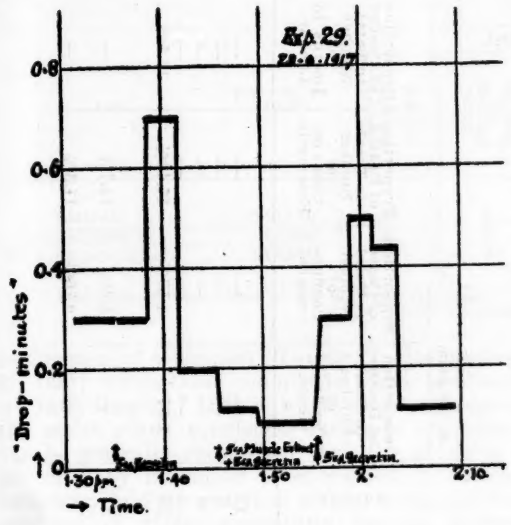
The toxin or causative element of eclampsia, whatever its origin, has some relationship to the products of conception, but not necessarily the fœtus, for there are cases on record in which eclampsia has developed in patients with hydatid mole in the absence of a fœtus;[35] the fœtus is certainly absent in cases of *post partum* eclampsia. On the other hand it has been shown that very often the fits and other symptoms of eclampsia cease when the fœtus *in utero* dies, even when not born.

There are other effects of presumable toxæmia. These include certain psychoses, hallucinations, insanity, mania. We know these occur in other intoxications such as typhoid fever or sepsis, but on the other hand they may be due to some endocrine aberration comparable to *dementia præcox* or climacteric insanity. Peripheral neuritis, headaches, impetigo, herpes and other skin diseases, chorea, the asthma of pregnancy, probably nervous in origin and possibly reflex similar to the asthma encountered in some patients with appendicitis, are also examples. I would also include the cases of unconsciousness that Williams[36] holds may have been cases of *encephalitis lethargica*, for we must not forget that the pregnant woman is liable to contract any intercurrent disease.

The glycosuria of pregnancy (which must not be confused with lactosuria from which it may be distinguished by the phenyl-hydrazine test) may be either alimentary, in which case diet will correct the condition, or toxic. My studies of these cases lead me to believe that we have here a toxic spoiling of the cells of the pancreas. I have found real benefit in these cases by giving secretin or secretogen three times a day one half-hour before food. By being absorbed from the duodenum before the ingestion of food it stimulates the secreting cells of the pancreas to increased effort with consequent hyperæmia from which the internal secreting apparatus also derives benefit. That toxins injected into the blood do inhibit the flow of pancreatic juice can be seen on reference to Graphs I. and II.

A condition which is not usually spoken of as a toxæmia, but whose toxic relationships deserve our earnest consideration, is accidental hæmorrhage, more especially concealed accidental hæmorrhage. When one sees how the uterine muscle is rotten, permeated with hæmorrhages and often irresponsive to any form of stimulus, chemical, thermal or mechanical, one is forced to wonder whether there is not some other factor at play besides merely hæmorrhage between the placenta and uterus, especially when one considers how these patients sometimes die quite suddenly even when the hæmorrhage has apparently stopped.

Finally as to the pathogenesis of the various conditions dealt with above. We are constantly confronted with examples of toxic spoiling of cells of the more highly evolved tissues, more especially the tubule cells in the kidneys of toxæmic nephritis, in acute yellow atrophy and in eclampsia and the liver cells in acute yellow atrophy and in eclampsia. Why the necrosis should start in the region of the hepatic veins in the former and the portal system in the latter is difficult to say. An attempt to supply the reason would only lead us in our present knowledge into the realms of speculation. It cannot be doubted that the increased flow of saliva met with in excessive salivation is due to the irritation of the cells of the salivary glands by the toxin. That such a condition can be produced experimentally, as is seen in Graphs I. and II., certainly supports such a contention. Whether the excess of mucus in the stomach in vomiting of pregnancy and in the urine in some cases of eclampsia and "pregnancy kidney" belong to the same category cannot be definitely stated at present, but I believe these conditions as well as the gastrostaxis of pernicious vomiting belong to the same category. It has been shown by Widdows[37] that the calcium content of the blood tends to decrease in the last months of pregnancy and to rise directly after confinement. We do know in a general way that calcium will neutralize the effects of certain toxins. The appearance of the stomach and bowel after neutral potassium oxalate has been administered by the œsophagus, thus depriving both of calcium, certainly suggests some relationship to the gastrostaxis of pernicious



GRAPH I.
"Drop-minutes" = the average number of drops per minute, being the inverse of the number of minutes taken for a drop to form.

vomiting. Of course the vomiting may also be due to irritation of the vomiting centre.

As regards eclampsia the headache, vertigo, *tinnitus aurium*, entoptic phenomena and convulsions suggest irritation of various centres of the brain, while the blindness, unconsciousness and coma suggest a deadening of these same centres. The stertorous breathing, full bounding pulse and raised blood pressure suggest irritation of the bulbar centres, although some of the hyperpiesis may be due to peripheral action of the toxin. It has been suggested that the entoptic phenomena and eye symptoms are due to pressure of a hypertrophied pituitary gland on the optic chiasma. The fact that even when the patient is blind an active pupillary response to light is still obtained and the reaction is still present, suggests a cerebral origin for the blindness. The epigastric sensation, I believe, is due to the toxic inhibition of the flow of pancreatic juice. Acid chyme then enters the duodenum; there is no alkaline pancreatic juice, *succus entericus* or bile to neutralize it and the duodenum is irritated and must in time be ulcerated. Such ulceration I have been able to produce in dogs by tying the pancreatic duct or by repeated subcutaneous injections of toxic material.[2]

As regards the predisposing causes of these toxæmias practically nothing is known. These causes must be studied in order to prevent the occurrence of the toxæmias. I would, however, like to point out that contrary to common belief the sweat does not contain toxins and probably toxins are not excreted by the skin (Van Noorden)[38] and also that diphtheria and tetanus toxin when injected into guinea-pigs are excreted in the urine, 50% of the toxin injected being recovered from the urine, while none at all is recovered from the bowel contents.[39] A few years ago I collected the statis-

TABLE I.

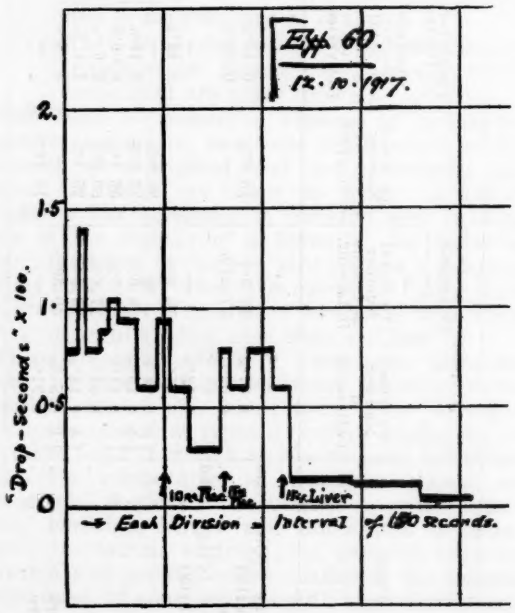
Place.	Proportion of Deaths from Eclampsia. To Total Number of Deliveries.	Proportion of Deaths from Eclampsia. To Total Number of Deaths in Labour.	Proportion of Deaths from Eclampsia. To Total Number of Cases of Eclampsia.	Proportion of Deaths in Confinement to Total Number of Deliveries.	Latitude.	Longitude.	Mean Temperature. Average (F.).	Mean Temperature. Minimum (F.).	Mean Temperature. Maximum (F.).	Mean Relative Humidity.	Total Rainfall (Inches).	Number of Rainy Days.	Wind. Mean Prevailing.	Wind. Mean Velocity (Miles per Hour).
Melbourne (City and Suburbs)	0.07%	15.0%	15.0%	0.46%	37° 50′ S.	144° 59′ E.	58°	67°	49°	71%	25.51	155.6	—	—
Adelaide	0.09%	27.27%	—	0.35%	35° 57′ S.	138° 38′ E.	63°	73°	53°	56%	21.04	110.4	—	—
Sydney (City and Suburbs)	0.12%	—	—	—	33° 52′ S.	151° 12′ E.	63°	70°	56°	73%	48.00	155.4	N.54°W.	3.5
Perth	0.07%	15.6%	—	0.44%	32° 0′ S.	115° 50′ E.	64°	73°	55°	62%	33.11	127	—	—
Bombay	a 0.09%	—	90.0%	—	18° 55′ N.	72° 54′ E.	79.3°	90.7°	67.9°	78%	73.99	—	N.62°W.	11.8
	b 0.19%	6.0%	23.5%	3.16%										
	c 0.11%	2.3%	56.8%	5.06%										
Delhi	0.63%	17.6%	60.0%	3.6 %	28° 30′ N.	77° 15′ E.	77.1°	104.7°	47.9°	54.9%	27.7	—	—	—
New York	a 0.18%	15.8%	35.4%	1.1 %	40° 45′ N.	74° 0′ W.	52°	82°	24°	73%	44.8	—	N.W.	—
Baltimore	b 0.5 %	40.66%	—	1.24%	39° 10′ N.	76° 30′ W.	55°	86°	28°	70%	43.4	—	S.E.	—
	0.91%	—	18.18%	—									—	—

tics of eclampsia of various countries, as well as obtaining their definitions of eclampsia. These figures were obtained at a time when the systematic antenatal treatment and observation as we now carry out were practically unattended to, so that the statistics are more or less comparable.

It will be seen from Table I. that in two cities of the same country wide differences are obtained, such as Bombay and Delhi, New York and Baltimore and in a lesser degree Melbourne and Sydney. The general geographical and meteorological conditions are more or less identical, so that the differences must be due either to some specially local condition, such as moisture in the soil, or to the effect on the body functions, of the differences in the characters of the prevailing winds and the rainfall. The more recent records for the women's hospitals of Melbourne and Sydney are appended (see Table II.).

That the incidence of eclampsia is apparently not closely connected with the incidence of nephritis is shown in the following statistics obtained from the records of the Women's Hospital, Melbourne (eclampsia) and from the Melbourne Hospital (nephritis), although in places the figures do approximate. The suburban areas of Melbourne are divided for the purposes of comparison into the following areas: Littoral, altitude under thirty feet above sea level; sublittoral, altitude under one hundred feet above sea level and outer-higher, altitude over one hundred feet above sea level.

It therefore appears that such conditions as predispose to the evaporation of water from the body as would exist when the body is exposed to heat and dry winds, apparently also predispose to the



GRAPH II.

"Drop-seconds" = the average number of drops per second, being the inverse of the number of seconds taken for a drop to form.

TABLE II.

Hospital.	Year.	Number of Confinements.	Eclampsia. Number of Deaths.	Proportion of Deaths to Total Number Confinements.	Eclampsia. Number of Cases. Onset. Ante and Intra Partum.	Onset. Post Partum.	Deaths.	Proportion of Deaths to Total Number of Confinements.	Proportion of Cases to Total Number of Confinements.	Proportion of Deaths to Total Number of Cases of Eclampsia.	Proportion of Deaths to Total Number of Deaths in Labour.
Women's Hospital, Melbourne	1921	2,080	36	—	35	3	13	—	—	—	—
	1922	2,033	43	—	21	3	3	—	—	—	—
	1923	2,130	28	—	28	3	4	—	—	—	—
	1924	2,172	32	—	32	7	3	—	—	—	—
	1921-1924	8,415	139	1.65%	116	16	23	0.27%	1.57%	17.42%	16.5%
Royal Hospital for Women, Paddington, Sydney	1920-1924	7,914	—	0.15%	—	—	12	—	1.3%	11.7%	—
Women's Hospital, Crown Street, Sydney	1920-1924	5,813	—	0.09%	—	—	5	—	0.76%	11.3%	—
					Number of Cases.						
Melbourne Hospital	1921	—	—	—	38	—	—	—	—	—	—
	1922	—	—	—	24	—	—	—	—	—	—
	1923	—	—	—	31	—	—	—	—	—	—
	1924	—	—	—	39	—	—	—	—	—	—
	1921-1924	—	—	—	132	—	—	—	—	—	—

TABLE III.

Area.	Population (1917).	Eclampsia.[1]				Nephritis.[2]			
		Total Number of Cases.	Total Number of Deaths.	Proportion of Cases.[3]	Proportion of Deaths.[3]	Total Number of Cases.	Total Number of Deaths.	Proportion of Cases.[3]	Proportion of Deaths.[3]
I.—Littoral	140,000	18	6	4.28	1.1	132	10	18.8	1.4
II.—Sublittoral	262,000	45	5	5.7	0.63	299	29	22.8	2.2
III.—Outer-higher ..	276,000	34	9	4.1	1.07	246	26	17.8	2.1

[1] Statistics of three years at the Women's Hospital. [2] Statistics of five years at the Melbourne Hospital.
[3] Per 100,000 of population *per annum*.

development of eclampsia (Tweedy[40] also mentions this fact) while those conditions which predispose to "water-logging," that is warm moist atmosphere with moist winds, predispose rather to nephritis.

It is generally recognized that the drinking of sea water by marooned or shipwrecked travellers induces irritability and then mania, while explorers *perishing* of thirst apparently become delirious *before* they die. I have attempted to elucidate experimentally whether desiccation of the body or raising the osmotic pressure of the blood increases the sensitiveness of the nervous system to convulsive drugs. It may be of interest here to mention that Oppenheimer[41] states that in pregnancy the osmotic pressure of the blood is higher than normal. I kept some rabbits in a warm room for some days without water to drink and I found that it required a smaller dose of strychnine to induce convulsions in these animals than in the normal. I have also injected concentrated Ringer's solution or saline solution intravenously into animals and the results so far obtained suggest that these animals become more readily convulsed by strychnine than the normal.

It may be of interest here to note how quickly (within five minutes) the blood of a dog whose total blood was about 750 cubic centimetres, injected with 220 cubic centimetres of Ringer's solution ten times normal strength became normal.

Experiment 137, November 22. 1920.

A dog was cannulized in the left carotid artery, the right jugular vein and the right femoral artery. At 3.40 p.m. a sample was taken from the femoral artery.

The refractive index of water was 1.333 and of serum 1.344.

From 3.41 to 3.52 p.m. 200 cubic centimetres of ten times concentrated Ringer's solution together with 7% gum acacia at 40° C. were injected.

At 3.59 p.m. the refractive index of the serum was 1.346.

At 4.10 the animal collapsed and had continuous convulsions.

Experiment 139, November 24, 1920.

A dog was prepared as in Experiment 137.

The refractive index of water was 1.3335. With concentrated Ringer's solution and 7% gum acacia it was 1.3485.

At 2.4 p.m. 220 cubic centimetres of ten times concentrated Ringer's solution were injected. The refractive index of the serum at 2 p.m. was 1.3430 and at 2.4 1.3462. At 2.10 0.6 milligramme of strychnine was injected and at 2.12 the refractive index was 1.3436. At 2.15 a further dose of 0.6 milligramme of strychnine was injected. The refractive index at 2.20 was 1.3436. At 2.21 twenty cubic centimetres of a 10% solution of calcium chloride were injected. At 2.25 the refractive index was 1.3431. From 2.30 to 2.33 220 cubic centimetres of water were injected and at 2.38 the refractive index was 1.3436. At 2.45 twenty-five cubic centimetres of magnesium sulphate were injected and at 2.50 the refractive index was 1.3450. The dog had convulsions after the second injection of strychnine.

It is also of interest to note that wherever there is a tendency to increased intraabdominal tension, such as in *primiparæ*, or patients with hydramnios or with multiple pregnancy, there is a tendency to eclampsia. I have attempted to imitate this condition by introducing in the abdominal cavity of the anæsthetized dog a small football bladder which can be inflated or deflated at will. The results so far obtained seem to indicate that the animal is more responsive to strychnine when the bladder is blown up than when it is deflated.

Treatment of Toxæmias.

Finally, may I be permitted to trench a little on what other lecturers will deal with, namely methods of treatment from their physiological aspect.

As regards bleeding this is found clinically of value where the blood pressure permits.

The following experiment is of interest.

Experiment 141.

To a large dog 2.4 milligrammes ($^1/_{25}$ grain) of strychnine sulphate were given intravenously; seven convulsions resulted. It was then bled of five hundred cubic centimetres into 0.2% solution of potassium citrate. The convulsions stopped.

This blood was centrifuged, the supernatant decanted off and saline solution with gum acacia added to the corpuscles. This suspension was run into the animal, but no convulsions followed.

It is apparent from this that the corpuscles do not carry the poison (strychnine) in these circumstances and it certainly suggests an improvement in the method of simple bleeding.

Drugs.

A few words regarding drugs.

Chloral hydrate[42] depresses sensory impressions and the motor areas of the cerebrum become less excitable to electrical stimuli. The tone of muscles generally is diminished and they relax. The heart dilates and the tone of the muscle coats of the arteries and arterioles is depressed.

Morphine[43] cuts off sensory impressions and therefore depresses reflexes. It acts on the sensory

cells of the nervous system. "Veratrone"[44] produces a slowing of the pulse by stimulating action on the cardio-inhibitory centre in the bulb and hence produces a fall in blood pressure. It also acts on striated muscle causing a very considerable prolongation of the period of relaxation after a contraction. Bromides and the like are obviously cerebral depressants.

In regard to the treatment of vomiting of pregnancy, "Luminal" is a depressant, while *corpus luteum* is an attempt at organo-therapy and is intended to replace a missing hormone, but this defect has never been satisfactorily proved by experiment. As regards salivation X ray treatment of the salivary glands has been recommended for the purpose of causing their degeneration. It seems a very drastic treatment for such a trifling and temporary condition and, as De Lee says,[45] it is like using cannon to shoot sparrows.

Prophylaxis.

Regarding prophylaxis, I can only suggest general hygienic measures and antenatal care with treatment of albuminuria, acetonuria, glycosuria and biliuria on the usual lines as soon as they appear. I have always advocated copious draughts of water or watery liquids, thus insuring efficient diuresis and preventing any tendency to "drying." I have been singularly fortunate, for in my last thousand confinements in private practice I have only had one patient with eclampsia and she admitted to me afterwards that she had not carried out my instructions regarding drinking of plenty of water. I may again mention that Tweedy in the Rotunda Reports of 1910 mentions deficiency of water as a predisposing cause of eclampsia.

In conclusion I would like to say that whatever our views may be as to the ætiology of the toxæmias of pregnancy and whatever treatment we may recommend for the various conditions, we must all be agreed as to the great value of antenatal observation and treatment and until the toxin or toxins responsible have been isolated, when prophylaxis and treatment should be easy matters, it is incumbent on us to use every method or treatment which experience or statistics prove to be of value in preventing these conditions.

References.

(1) Whitridge Williams, "Obstetrics," 1912, page 560.

(2) Claude Bernard, "Paralytic Secretion of Salivary Glands," *Journal de l'anatomie et de Physiologie, Tome* I., 1864, page 507 (quoted by Schäfer, "Textbook of Physiology," Volume I., 1898, page 519).

(3) J. Leon Jona, "An Experimental Study of Duodenal Ulcer," THE MEDICAL JOURNAL OF AUSTRALIA, March 2, 1918, page 143.

(4) Whitridge Williams, *loco citato*, page 519.

(5) Comyns Berkeley and T. I. Bennett, "The Toxæmias of Pregnancy," *The Lancet*, Extra Numbers, No. I., "Modern Methods in Abnormal and Difficult Labour," 1924, page 6.

(6) Whitridge Williams, *loco citato*, page 520.

(7) Whitridge Williams, *loco citato*, page 522.

(8) J. Leon Jona, "A Further Contribution to the Experimental Study of Fever; II. Burns," THE MEDICAL JOURNAL OF AUSTRALIA, November 3, 1917, page 373.

(9) E. Speidel, "A Routine Treatment for Hyperemesis Gravidarum," *American Journal of Obstetrics and Gynecology*, 1923; page 481 (Abstract in *Surgery, Gynecology and Obstetrics*, September, 1923, page 254).

(10) A. R. Rich, "On the Extrahepatic Formation of Bile Pigments," *Johns Hopkins Hospital Bulletin*, April, 1925, page 257.

(11) Bertillon System of Nomenclature (Nomenclature of Diseases agreed upon by the International Commission on the International Nosological Nomenclature in Paris), 1909 (Commonwealth of Australia Bureau of Census and Statistics, 1910, page 146).

(12) "Rotunda Definition of Eclampsia," *Rotunda Hospital Reports*, 1910, page 4.

(13) Whitridge Williams, *loco citato*, page 546.

(14) Whitridge Williams, *loco citato*, page 537.

(15) New Zealand Government Maternity Hospitals, Ten Years, 1906-1915. Information supplied by personal letter from the District Health Officer, Auckland, August, 1916.

(16) New South Wales Subsidized Hospitals in the Metropolis, Information supplied by personal letter from the Medical Officer of Health of Sydney, August, 1916.

(17) Cassamajor, quoted by Whitridge Williams, *loco citato*, page 537.

(18) R. H. Morrison, "Eclampsia," Transactions of the Australasian Medical Congress, Sydney, 1911, Volume I., page 409.

(19) E. Zweifel, quoted by Sajous, "Cyclopedia of Internal Medicine" Volume VII., page 706.

(20) Whitridge Williams, *loco citato*, page 538.

(21) E. A. Schäfer, "Textbook of Physiology," 1900, Volume II., page 741.

(22) Whitridge Williams, *loco citato*, page 555.

(23) Lichtenstein, *Zentralblatt für Gynäkologie*, 1909, *Seite* 1313; *Archiv für Gynäkologie, Band* XCV., 1911, *Seite* 183.

(24) Gibbon Fitzgibbon, "Eclampsia: Including the Pre-eclamptic State," *The Lancet*, Extra Numbers, No. I., "Modern Methods in Abnormal and Difficult Labour," 1924, page 121.

(25) J. Leon Jona, "A Contribution to the Experimental Study of Fever," *The Journal of Hygiene*, January 14, 1916, page 182.

(26) Whitridge Williams, *loco citato*, page 542.

(27) Whitridge Williams, *loco citato*, page 540.

(28) Neubauer-Huppert, "*Analyse des Harns*," 1910, *Seite* 236.

(29) Levant and L. Portes, "*Hémorragies des Centres Nerveux au cours de l'éclampsie Puéperale*," *Gynécologie et Obstétrique*, 1923, *Tome* VII., *page* 332 (Abstract in *Surgery, Gynecology and Obstetrics*, September, 1923, page 473).

(30) C. S. Wallace, H. H. Dale, P. P. Laidlaw, A. N. Richards, W. M. Bayliss, W. B. Cannon and others, "Traumatic Toxæmia as a Factor in Shock," Medical Research Committee, National Health Insurance, Special Report Series, No. 26, 1919.

(31) Whitridge Williams, *loco citato*, page 548.

(32) J. E. Talbot, "Toxæmias of Pregnancy from the Standpoint of Chronic Sepsis as Ætiological Factor," *American Journal of Obstetrics and Gynecology*, December, 1923.

(33) F. von Bemuth und F. Goebel, *Biochemische Zeitschrift*, 1924, Nr. 146, *Seite* 336 (quoted in *Physiological Abstracts*, Volume IX., August, 1924, page 233).

(34) A. Mahnert, *Archiv für Gynäkologie, Band* CXIX., *Seite* 407 (Abstract in *Surgery, Gynecology and Obstetrics*, June, 1924, page 534).

(35) Falk and Litzenfrey (quoted by Sajous, "Cyclopedia of Internal Medicine," Volume VII.).

(36) Whitridge Williams, *loco citato*, page 561.

(37) S. T. Widdows, "Calcium Content of the Blood During Pregnancy," *Biochemical Journal*, Volume XVII., 1923, page 34.

(38) Van Noorden, "Pathology of Metabolism," Volume II., page 510.

(39) R. Bieling and A. Gottschalk, *Zeitschrift für Hygiene und Infektionshrankheiten*, 1923, *Seite* 142 (Abstract in *Medical Science*, Abstracts and Reviews, Volume IX., February, 1924).

(40) J. Tweedy, *Rotunda Hospital Reports*, 1910.

(41) Oppenheimer, "Physiological Chemistry," Volume II., Part 2, page 4.

(42) A. C. H. Rothera, "Pharmacology," Second Edition, page 37.
(43) A. C. H. Rothera, "Pharmacology," Third Edition, page 39.
(44) C. E. de M. Sajous, "Cyclopedia of Internal Medicine," Volume VIII., page 805.
(45) De Lee, "Obstetrics," Practical Medicine Series, 1924, page 286.

Reports of Cases.

INFECTIOUS MONONUCLEOSIS OR GLANDULAR FEVER.[1]

By J. Gordon Hislop, M.B., Ch.B. (Melbourne) M.R.C.P. (London),
Medical Superintendent, Children's Hospital, Perth.

Clinical Histories.

Case I.

On July 17, 1925 a female child aged ten years, was sent to the out-patient department of the Children's Hospital with a request for admission on account of tuberculous glands of the neck.

The following history was obtained from the parent:

The child seemed perfectly well up till seven days previously, when she became languid and listless and refused to take her food, losing her usual good colour. A slight cough had been noticed for four or five days only. No history of vomiting or any hæmorrhages was given. Constipation had been prominent.

On examination the child appeared pale, but did not look ill. The cervical glands were enlarged bilaterally in both anterior and posterior triangles, they were discrete, not tender, not unduly hard, the enlargement being moderate in degree. To a lesser extent the glands in both axillæ and inguinal regions were enlarged and of the same nature.

The teeth were in good order. The tongue was slightly coated. The tonsils were a little enlarged, but not obviously infected. The heart was clear and no abnormality was to be detected in the lungs. The liver was enlarged to about two finger's breadth below the costal margin, the edge being quite hard to the touch. The spleen was enlarged to more than one finger's breadth below the costal margin and was tender on pressure.

The patient was admitted to a medical ward with the provisional diagnosis of (?) glandular fever, (?) Hodgkin's disease. A blood examination on July 20, 1925 gave the following result: Red blood cells 5,100,000 per cubic millimetre, hæmoglobin value 60%, leucocytes 18,125 per cubic millimetre.

The relative count was as follows: Polymorphonuclear leucocytes 12.6%, lymphocytes 83.6%, large mononuclear leucocytes 2.4%, eosinophile cells 1.4%. No reaction occurred to the von Pirquet test.

On July 28, 1925 the spleen had diminished in size until it was just palpable, the liver remaining unaltered in size, whilst the cervical glands had become much smaller.

On August 3, 1925 the total leucocyte count had fallen to 14,060 per cubic millimetre. The relative count was as follows: Polymorphonuclear leucocytes 45%, lymphocytes 52.5%, large mononuclear leucocytes 1.5%, eosinophile cells 1.0%.

On August 12, 1925 the spleen was no longer palpable, the liver being palpable just below the costal margin.

On August 15, 1925 the child developed influenza and the glands which had returned to almost their normal size, increased again slightly.

On August 18, 1925 the total leucocyte count had fallen to 9,000 cells per cubic millimetre. The differential count was as follows: Polymorphonuclear leucocytes 55%, lymphocytes 43.5%, large mononuclear leucocytes 1.5%.

Case II.

This patient (T.H.E.), was a child of one year and eight months, who was also sent to hospital with a diagnosis of tuberculous adenitis on August 5, 1925. The mother stated that the child had developed a cold a few days previously. The glands in anterior and posterior triangles were moderately enlarged, shotty and discrete. Some enlargement of the inguinal glands was noted. The liver was slightly enlarged, being definitely palpable and whilst the spleen was not palpated, there was distinct dulness over the splenic area.

A blood examination gave the following results: Red blood cells 4,106,500 per cubic millimetre, leucocytes 6,250 per cubic millimetre, The differential count was as follows: Polymorphonuclear leucocytes 13%, lymphocytes 84%, large mononuclear leucocytes 2.4%, eosinophile cells 0.6%.

Case III.

H. V., a female, aged five years, had become sick six weeks previously. She complained of aching with some swellings in the neck. She was vomiting and feverish at the onset. She improved after one week, but had not seemed well since, did not eat well and had a cough.

On examination the glands were seen to be enlarged in both posterior triangles of the neck, shotty, discrete The spleen was not palpated. The liver was just palpable.

The patient's condition was investigated hæmatologically for the reason that she had been playing with patient No. I. and to determine how long the blood condition would persist. On the history this may have been the primary case. Examination of the blood gave the following result: Red blood cells 3,000,000 per cubic millimetre, leucocytes 9,050 per cubic millimetre. The differential count was as follows: polymorphonuclear leucocytes 34.9%, lymphocytes 61.5%, eosinophile cells 3.6%.

Whilst there may have been some hesitation in accepting this examination as evidence, the child is old enough to have grown beyond the infantile tendency to develope lymphocytosis in infectious conditions.

From the age of five onwards there is little difference in the response to infection of the child or adult.

Case IV.

The fourth patient was a nurse who had first complained of a glandular enlargement of the left cervical region, the temperature being 38.9° C. (102° F.). The tonsils presented a few follicular spots, but by the third day the glands in the anterior and posterior triangles were enlarged and the patient complained of stiffness of the neck. By the fifth day the spleen and liver were both palpable and the axillary and inguinal glands were enlarged and tender, though as elsewhere they remained discrete without any redness of the skin over them. On this same day the temperature fell to normal, but a blood count gave the total leucocytes as 4,250 per cubic millimetre and the percentage of lymphocytes as 92. By the fourteenth day the spleen was no longer palpable and a blood count showed the polymorphonuclear leucocytes as being relatively equal in number to the lymphocytes. A month elapsed before the glands finally subsided and three months later blood examination gave a normal result.

Comment.

In 1886 Filatow, of Moscow, first described the disease as glandular inflammation of the neck without inflammatory changes in the mouth, nose or pharynx.

Pfeiffer in 1889 gave the first clear account of the disease.[(1)] Dawson Williams in 1897 also gave a clear account of the symptomatology,[(2)] but it is only the later writers who describe the accompanying blood changes which gave the disease its new terminology, infectious mononucleosis.

Ætiology.

The cause is obscure. Comby[(3)] and Ashby and Wright[(4)] suggest that the infection is an attenuated form of streptococcus, but Dawson Williams doubted this, holding

[1] Read at a meeting of the Western Australian Branch of the British Medical Association on August 19, 1925.

the view that they had based their opinion on the fact that Neumann had isolated staphylococci in some glands which suppurated. Most writers remark on the absence of suppuration. The presence of constipation which all observers have noted, *plus* the fact that the left side is usually first affected has lead to the theory of intestinal origin through lymphatic channels. Whilst many agree that the point of entry is possibly through the tonsil or pharynx, the glands are not as a rule affected in the same order as they are following tonsillar infection.

Whilst most of the reported cases occur in children from five to fifteeen years of age, mild epidemics have been reported in which young adults have been affected.

Pathology.

Leucocytes may range as high as 26,000 per cubic millimetre though one observer maintains that there is a leucopenia, along with an absolute and relative increase in the non-granular mononuclear cells.

Longcope[5] states that 80% to 90% of the leucocytes are of non-granular type and appear as: (i.) A small mononuclear leucocyte identical with the small lymphocyte seen in normal blood; (ii.) large mononuclear cells identical in appearance with the large mononuclear and transitional cells of the normal blood; (iii.) mononuclear cells of a type not usually encountered in normal blood. The cells of this last group are larger than small lymphocytes. They contain oval, kidney shaped, slightly lobulated or Riedel type nuclei, staining deeply with Wright's and Hasting's stain. The nucleus is large and often surrounded by non-granular cytoplasm. They do not give the oxidase reaction and are therefore not of myeloid origin. After a few days the leucocytosis clears up and the mononuclear cells slowly disappear.

Symptoms.

Unfortunately, the early symptoms had abated when these patients, with the exception of No. 4, were admitted to hospital.

All observers agree regarding the acute onset with malaise, anorexia, obstinate constipation, pain and stiffness in the neck, abdominal pain and high fever with a corresponding rapid pulse rate.

The condition of the tonsils does not seem to be sufficient from clinical inspection to warrant a diagnosis of tonsillitis. The glandular enlargement occurs early, probably forty-eight hours after onset, the usual site of the first enlargement being just beneath and posterior to the left sterno-mastoid muscle. The glands then become enlarged in chains on both sides of the neck, anteriorly and posteriorly. The glands in the axilla and groin may be but need not be enlarged.

The acute symyptoms begin to abate about the fifth day and the glands to resolve. The liver is enlarged almost invariably and the spleen in about half the cases. The mediastinal and bronchial glands may be enlarged and possibly this accounts for the cough which so often accompanies the infection and persists for some time.

Prognosis.

The disease is rarely fatal, though it may be severe, the present cases quoted being of an apparently mild type. There is, however, some evidence to show a similarity between this infection and one known as epidemic sore throat which can be a serious infection. The blood changes in the latter condition are not very definitely reported.

Various estimates are given as to the length of time that the glands may take to resolve. Dawson Williams gives a fortnight, Kellert[6] gives up to one month and Box,[7] three weeks. The child is left in an anæmic condition for a month or two afterwards and deserves appropriate treatment.

Complications.

Acute nephritis is the most common complication and is said to occur in about 6% of the cases.

Treatment.

Various drugs have been recommended, proving that none are specific. Some state that treatment has little influence on the course of the disease and use this in support of their opinion that the disease resembles a specific infection.

Sodium iodide, hexamine, iron and arsenic are the commonly used drugs and in view of the anæmia left after the infection, the latter two seem specially indicated.

Differential Diagnosis.

Differential diagnosis is very difficult without a full blood examination.

The sudden onset with all the typical symptoms will render the diagnosis more simple, but on the mild case the blood picture and count are indispensable.

Tuberculosis, syphilis and Hodgkin's disease have not the sudden onset and are more chronic.

Acute leuchæmia may present some difficulty, but may be ruled out by the low leucocytosis or the leucopenia.

References.

(1) Emil Pfeiffer: *Drüsenfeiber, Jahrbuch für Kinderheilkunde*, 1889, *Band* XXIX., *Seite* 257.
(2) Dawson Williams: "A Note on the Glandular Fever of Childhood," *The Lancet*, Volume I., 1897, page 160.
(3) Comby: "La Médecine Infantile," *Tome* I., *page* 1.
(4) H. Ashby and G. A. Wright: "The Disease of Children, London," 1896, page 255.
(5) W. T. Longcope: "Infectious Mononucleosis," *American Journal of the Medical Sciences*, Volume CLXIV., 1922, page 781.
(6) Ellis Kellert: "Infectious Mononucleosis," "Nelsons Loose Leaf Living Medicine," Volume II., 1924, page 219.
(7) Charles R. Box: "Glandular Fever," "Text book of the Practice of Medicine," by various authors, edited by Frederick W. Price, 1922, page 582.

Reviews.

A TEXTBOOK OF ANATOMY.

THE University series of textbooks which includes many well known and standard manuals, is well represented in the field of anatomy by Buchanan's "Manual of Anatomy."[1] This book has now attained its fifth edition. It is representative of the London school of anatomy, being edited by Barclay-Smith, Frazer, Parsons and Wright, all London teachers. The chief claim to distinction and one which will appeal to senior graduates, is that it is the only standard textbook of anatomy which retains the "old" nomenclature. This, the fifth edition has been enlarged considerably, nearly two hundred new figures having been added. The practical guide to dissection has been omitted and more space has been given to descriptive text.

The section on embryology is clear and concise and is illustrated by many useful and simple diagrams. The description of the skeleton is not overloaded with detail and the necessary information on ossifications is well arranged. The description of the limbs, abdomen and head and neck is arranged regionally and not by systems. This is very convenient for those who wish to revise the anatomy of any particular part, but it does not give the broad systematic view of the structure of the body that is so necessary clinically. The central nervous system and the special senses have separate sections devoted to them.

It would add to the value of the descriptive section if the relations of the surfaces and borders of the chief muscles were indicated. This is always useful to the surgeon and student.

The book is well printed and illustrated and is well worthy of its place in the University Series.

[1] "Buchanan's Manual of Anatomy, Including Embryology," Edited by E. Barclay-Smith, M.D., J. E. Frazer, F. G. Parsons, F.R.C.S., W. Wright, F.R.C.S.; Fifth Edition; 1925. London: Baillière, Tindall & Cox. Demy 8vo., pp. viii. + 1,702, with 810 illustrations. Price: 35s. net.

The Medical Journal of Australia

SATURDAY, NOVEMBER 7, 1925.

The Tuberculosis Problem.

THE non-medical mind apparently has the greatest difficulty in taking a wide view in regard to the proper method of attacking preventible disease. The lessons of typhus fever and of variola are usually unheeded. And yet there is a great deal of eloquent evidence which proves that the discovery of a real curative agent of an infective process possesses a far smaller national significance than the application of the agent to the prevention of the disease. It has been possible to stamp out typhus fever and variola by scientific measures properly applied. When these diseases appear, it may be concluded that there has been a serious failure in the resort to these preventive measures. It has been said that the existence of leprosy in a community should be regarded in these days as a reproach to that community. The discovery by von Behring of antitoxin as a curative agent in diphtheria has not resulted in the elimination of death from that disease. The determination of susceptibility among those exposed to infection by the Schick test and the raising of the resistance in the susceptible are measures of much greater importance, since they can be applied thoroughly and at the proper time. By their means both the morbidity and the mortality of diphtheria can be combated.

There has been a persistent demand on the part of certain sections of the community for help to be extended by the Government to Monsieur Spahlinger in order that his serums and vaccines may be available in Australia. A year ago Sir David Hardie dealt with this subject in this journal and came to the conclusions that medical evidence in regard to the efficacy of this form of treatment of pulmonary tuberculosis was wanting, that at best it was still in the experimental stage and that Monsieur Spahlinger's claims were "non-proven." At the same time an attempt was made editorially to examine the probabilities connected with these claims. Unfortunately Monsieur Spahlinger has refused to do what every reputable member of the medical profession of the British Empire undertakes to do, namely to make known each detail of his experimental work. This mystery naturally created an atmosphere of suspicion. Monsieur Spahlinger has been asked on scores of occasions to permit his methods to be controlled and tested and he has on each occasion refused to supply the information needed. When a medical practitioner claims to have discovered a remedy capable of curing such a deadly disease as tuberculosis, he publishes the details of his experimental work and gives a full account of the method followed in the preparation of the remedy. It is expected of him that he will be frank with all scientific critics and permit them to see all he has to show in his laboratory. When a layman claims that he has made the same discovery, the medical profession looks askance if no details are vouchsafed. Monsieur Spahlinger has attracted public attention for a long time. He has not been frank and is not willing to publish an account of his methods. The fact that a few clinicians who have no experience of laboratory work, have been persuaded to accept his claims on very poor evidence, is not sufficient reason to induce the medical profession as a whole to trust him. We have pointed out that many investigators have acclaimed the discovery of a specific treatment for tuberculosis. Since Koch gave to the world his tuberculin, hundreds of others have sought to improve on the master's methods. Today Koch's tuberculin is still the best vaccine. There is no information to hand to persuade us that Spahlinger's vaccine is even an improved tuberculin. We have learned from time to time of sera which are supposed to have a curative action in this disease. If medical workers with great reputations as scientists have erred through excessive enthusiasm in this direction, why should we believe that this layman who keeps his methods secret, has succeeded? From the clinical records of persons treated by Monsieur Spahlinger's remedies we gather that some have improved, some have not improved and some have gone steadily downhill. The same occurs when no specific treatment is employed. In other words there is not a tittle of

evidence in favour of his remedies from the clinical point of view. In these circumstances we maintain that public money should not be wasted in supporting the manufacture of remedies the value of which is probably non-existent.

Current Comment.

INFECTIOUS MONONUCLEOSIS.

Comparatively little has been written in textbooks about infectious mononucleosis and its ætiology has not been definitely determined. The four cases reported by Dr. Hislop in this issue would make it appear, however, that the condition is not one of great rarity. As Dr. Hislop points out the symptoms are fairly characteristic and after careful perusal of his statements it seems that if a full and careful blood examination is made, the diagnosis should not be a matter of extreme difficulty. That the diagnosis is not always a straightforward matter has recently been shown by Dr. J. F. Landon.[1] Dr. Landon has pointed out that although the blood of a patient suffering from acute leuchæmia may contain a preponderating number of small lymphocytes, the smear may manifest a relative and absolute increase of the pathologically large mononuclear cells. It is in this type of picture that confusion and uncertainty of diagnosis arise. Dr. Landon also points out that the so called aleuchæmic leuchæmia in which the total leucocyte count is normal or subnormal, adds to the confusion. On account of the uniformly bad prognosis in leuchæmia he regards it as quite possible that the condition in patients reported as having recovered from acute leuchæmia was in reality infectious mononucleosis.

Two cases reported by Dr. Landon are of interest when considered in conjunction with Dr. Hislop's article. The first patient was a woman, aged twenty-six years. She became ill with what was to all intents and purposes a mild pharyngitis. She did not recover as quickly as was expected and a blood examination was made. It was found that the white cells numbered 12,500 per cubic millimetre and that 86% of these were mononuclear cells (78% large mononuclear cells and 8% small lymphocytes). At a second examination the white cells numbered 15,000. The majority of the mononuclear cells were distinctly abnormal, the predominating type was somewhat larger than the larger lymphocytes, the cytoplasm stained lightly and the oval, lobulated or kidney shaped nucleus stained intensely. The cells did not give the oxidase reaction. Subsequently the patient's condition became much worse and a membrane apparently due to Vincent's angina spirillum formed on the tonsils. This diagnosis was confirmed in the laboratory. After treatment with intravenous injections of "Arsphenamin" the condition of the patient rapidly improved and the spleen, previously palpable, became reduced to a normal size. Six weeks after the onset of the illness the leucocytes numbered 6,400 per cubic millimetre, but 58% of them were mononuclear cells. The patient's condition was regarded as being undoubtedly due to infectious mononucleosis.

An interesting point arises in connexion with this case in regard to ætiology. Dr. Hislop refers to the view that the infection is due to a streptococcus and to the hypothesis that the path of infection is by the intestinal tract. Dr. Landon mentions that some writers have considered infectious mononucleosis to be a form of infection with the spirillum of Vincent. The case reported by him lends support to that view. At the same time he refers to the work of Tarnow who studied the blood of persons suffering from Vincent's angina. Tarnow found that the mononuclear cells in the blood of these individuals ranged from 5.6% to 27.6%. Dr. Landon, however, draws attention to the fact that many persons suffering from Vincent's angina manifest no abnormality in the blood. He holds that infectious mononucleosis is not a peculiar individual response to ordinary infections and gives as his reason the findings reported by Sprunt and Evans. These two observers found a typical polymorphonuclear response to ordinary pyogenic infection in a patient who had suffered from acute mononucleosis twelve months previously.

The second case reported by Dr. Landon is of importance from the diagnostic standpoint. The patient, a girl aged sixteen years, complained of pain in the upper part of the abdomen and of sore throat. She manifested a follicular tonsillitis. With the exception of an enlarged gland in the hilus of the left lung no abnormality was found on clinical and radiological examination of the chest. Remarkable findings resulted from examination of the blood. The white cells varied in number from 8,400 to 36,800 per cubic millimetre and the percentage of mononuclear cells varied from 80% to 96%. At *post mortem* examination miliary tuberculosis was found in the lungs, liver and spleen. No pronounced abnormality could be found on examination of the bone marrow. Before death a diagnosis of acute lymphatic leuchæmia was made. Dr. Landon can find only one similar case in the literature and it was reported by Wiechmann. In this instance the mononuclear cells represented 64% of the leucocytes.

These reports by Dr. Hislop and Dr. Landon apart from the interest of the case reports themselves teach several things. In the first place every available means of examining the patient, clinical, laboratory, radiological and so forth, must be used. Secondly even when this has been done, the nature of the condition may not be evident and it will be necessary to await the development of the course of the disease and possible repeated examinations of an exhaustive character will be required. Finally the importance of reporting in detail all cases of this description is evident, for only by much study will the nature of the condition be revealed.

[1] *The American Journal of the Medical Sciences*, July, 1925.

SYPHILIS OF THE CENTRAL NERVOUS SYSTEM.

But a few years ago the medical profession adopted an almost fatalistic attitude toward general paralysis of the insane and *tabes dorsalis*. It was thought that these conditions were parasyphilitic rather than syphilitic and that when the first manifestations of either disease appeared, it was useless to employ any specific remedies. By parasyphilitic was meant a condition appearing long after the direct effects of the syphilitic virus had developed. Many authorities believed that some chemical product attacked the tissue of the central nervous system. Fortunately this conception has proved to be without foundation. It is now known that both *locomotor ataxia* and general paralysis of the insane are caused by a direct attack by the *Spirochæta pallida*. It is probable that the cerebral centres and the posterior spinal nerve roots are involved much more readily when the metabolic and circulatory conditions of these areas are impaired. The impairment may be acquired or inherited. It is very difficult to reconcile the view that stress in itself causes a diminished resistance to the spirochæte with what is known concerning function of tissue cells and biochemical tissue change. It is far more probable that the acquired impairment of the circulatory integrity of the spinal or cerebral areas is occasioned by a localized vascular lesion. The arterial poisons may play a part in the selection of the site of the syphilitic attack. In these circumstances the prospect wears a more wholesome aspect. Notwithstanding the fact that the lesion in the central nervous system is a degenerative one, it is capable of arrest and even of repair, provided that the essential cause be destroyed or removed. With the knowledge that the spirochæte can be discovered in the central nervous tissue, clinicians began to adopt energetic antisyphilitic treatment in the early stages of both forms of neurosyphilis. The results were confusing. Some reports of undoubted improvement and even of apparent arrest heartened neurologists to increased effort. It was recognized, however, that care had to be exercised before a cure could be established, since symptomatic improvement is not uncommon after treatment by suggestion as has been demonstrated unconsciously by Mr. Hickson, the faith "healer." The real difficulty was found to lie in the obstacle offered by the chorioid plexus to the passage of spirochæticidal drugs. Hoefer endeavoured to show that if a full dose of an arseno-benzol drug were given and twenty to thirty minutes later, when the drug was still contained in the circulating blood, a considerable quantity of cerebro-spinal fluid were withdrawn by lumbar puncture, the drug would pass through the chorioid plexus into the cerebro-spinal fluid. The results of this treatment seemed to be better, but they were not so convincing that clinicians were justified in giving a good prognosis in neurosyphilis in its early stage.

The next observation of importance was that attacks of certain acute infective diseases were followed by distinct signs of improvement in *locomotor ataxia*. It was claimed that this improvement was progressive. This observation gave birth to the conception that neurosyphilis could be treated to advantage by inoculating the patient with the *Plasmodium* of malaria, allowing him to have a number of sharp attacks of fever and then treating the malaria with quinine. Here again the therapeutic measure was incomplete. Nature is often eager to rid the body of an invading organism, but demands the assistance of a somewhat complicated mechanism acting under favourable conditions. Artificially induced malaria alone apparently will not cure many patients of general paralysis or *tabes dorsalis*. Something more is required.

In 1915 Jacobs and Heidelberger, working at the Rockefeller Institute, prepared an arsenical drug, the sodium salt of N-phenyl-glycineamid-p-arsenic acid, and injected it into the muscles or veins in human beings and animals affected with trypanosomiasis. The results were said to be favourable. Later they tried the drug which they call "Tryparsamide," in general paralysis of the insane and *tabes dorsalis*. Excellent results were recorded. It is claimed that the spirochæticidal action of the drug is relatively feeble, but that it exercises a strong tonic action, that it promotes recovery from toxic injury and induces the healing of syphilitic lesions. The drug seems to have little general or local irritant action on the tissues. Dr. W. S. Dawson has obtained a supply of the drug and has employed it in the treatment of twenty patients, eleven of whom were suffering from general paralysis of the insane, six from tabo-paralysis and three from *tabes dorsalis*.[1] The disease in the majority of the patients was in an early stage and the mental symptoms were relatively mild. Each patient received eight injections of two grammes of "Tryparsamide" dissolved in from five to ten cubic centimetres of distilled water. Dr. Dawson states that he prefers intramuscular injections because the drug is eliminated from the system less rapidly than after intravenous injection. Details of various biological reactions are given, including the results of the Wassermann test applied to both the serum and the cerebro-spinal fluid, the cell and protein content of the spinal fluid and the result of Lange's gold sol test. The clinical data of twelve patients are also appended. He sums up his results as follows. Of the twenty patients thirteen were clinically improved, five were clinically not improved and two have died. Seven patients were "serologically improved" and eleven were "serologically not improved." It is interesting to note that two patients had been treated by inoculation with the blood of malarial persons. Neither of these patients was benefited by the febrile attacks or by the injections of "Tryparsamide." It has been determined that arsenic is present in the cerebro-spinal fluid for two or three days after the injections. Dr. Dawson realized that his observations are too few and too short to justify any definite conclusions concerning the value of this drug in the treatment of neurosyphilis, but he holds that they justify further and more extensive trials. With this all must agree.

[1] *The Lancet*, May 23, 1925.

Abstracts from Current Medical Literature.

OPHTHALMOLOGY.

Treatment of Trachoma with Strong Solution of Silver Nitrate.

N. R. DONNELL (*Archives of Ophthalmology*, September, 1924) has had twelve years' experience with trachoma in the south-east Missouri country, where the disease is rife and responsible for from 20% to 50% of blindness. Considering silver nitrate the best remedy he began using it in increasing strengths. He cocainized the eyes and massaged the everted lids with a wool-wrapped tooth pick dipped in a 2% to 5% and sometimes 10% solution of silver nitrate. Excess of fluid should be removed from the application and the conjunctival sac flushed out with salt solution. Ice packs lessen the subsequent discomfort. The treatment should be carried out by the ophthalmologist only and his experience should guide him as to the necessity for increasing or decreasing the strength of the solution. This method applies to the acute follicular and dermic cicatrical conditions; pannus and corneal ulcer are not contraindications to its use. Argyrosis need not be feared with a dry application, fresh solutions and proper flushing of the conjunctival sac.

Bismuth Salts in Ocular Tuberculosis.

D. GOURFEIN (*Revue Générale d'Ophtalmologie*, January, 1925) employed intramuscular injections of 0.20 gramme of tartrobismuthate of potassium and sodium in the treatment of several patients suffering from tuberculous iridocyclitis with remarkable success. Experiments with rabbits showed that treatment with injections of bismuth after inoculation with tubercle bacilli arrested the progress of the disease in all instances and cured it in some. Control rabbits inoculated and not treated progressed as a rule to general tuberculosis.

Percentage Evaluation of Macular Vision.

A. C. SNELL AND SCOTT STERLING (*Archives of Ophthalmology*, September, 1925) publish results of efforts to express the usual fraction denoting visual acuity as a percentage of visual efficiency. The fractions of the Snellen notation do not express fractional parts of macular vision. They are relative measurements of visual angles. They should not be reduced like other fractions nor be expressed as decimals. Clinical data show that no loss of earning ability was found until the acuity was less than $^{20}/_{60}$ in the better eye. An acuity of $^{20}/_{40}$ was found to cause no loss of earning ability and only a slight loss of efficiency. Acuity of $^{20}/_{400}$ was found to be a total incapacity in most cases. Experimentally glasses scratched with fine lines were employed to reduce vision. A glass sufficiently scratched to reduce vision to $^{20}/_{40}$ was standardized and used as a unit. Six of these glasses in series before the eye reduced vision to qualitative sight. From mathematical considerations it was shown that as the visual angle increases in arithmetical ratio, visual efficiency diminishes in geometrical ratio. Since six of the $^{20}/_{40}$ unit meshed glasses reduced vision to perception of light, the reduction caused by one of them ($^{20}/_{40}$) is approximately one-sixth of the whole—that is 16%. On this basis the loss of visual efficiency is approximately 50% for $^{20}/_{100}$ and 80% for $^{20}/_{200}$. A full table of corresponding values is given.

Electric Cataract.

W. S. FRANKLIN AND F. C. CORDES (*The Journal of the American Medical Association*, July 25, 1925) relate a case of cataract following electric shock and burn. The patient, a man of thirty-four years, came in contact with a live wire which caused a burn over the left eye and right breast and forearm. The voltage was two hundred and twenty. Nine months later he developed cataract in both eyes, more prominent in the left. Many authors have reported cataracts from commercial electricity. The voltage varies from two hundred and twenty to fifty thousand. The cataract may become evident within two years. It is characterized by flaky or granular opacities in the cortex and deeper layers. In industrial conditions this possibility should be noted.

Cyst of the Iris.

R. E. WRIGHT (*The British Journal of Ophthalmology*, September, 1925) describes a method of treating cysts of the iris which he used successfully in one instance. The cyst was filled with pure carbolic acid solution and emptied in the following manner: Two short syringe needles were introduced subconjunctivally into the cyst from opposite directions. One syringe was filled with normal saline solution and the other was empty. The empty syringe was used to aspirate the cyst, it was detached from its needle, emptied and a few minims of pure carbolic acid solution were taken up. This was introduced into the cyst and straightway aspirated. The other syringe was then operated filling the cyst with saline solution till washed free of carbolic acid solution.

Non-Operative Treatment of Heterophoria.

H. W. WOOTTON (*Archives of Ophthalmology*, September, 1925) says that it would be better to replace the term esophoria by its two causes, convergence excess and divergence insufficiency and the term exophoria by divergence excess and convergence insufficiency. Convergence excess (esophoria) is usually accompanied by hypermetropia and the treatment is convex lenses. Convergence insufficiency (exophoria) may be accompanied by myopia and is cured by concave glasses. If it is combined with hypermetropia, convex lenses may make it worse. It is best treated by increasing the converging power by exercising with prisms whose base is turned outwards, or when where reading glasses are required by combining a prism with the base inwards with the convex glass. Divergence excess (exophoria) requires operative treatment. Divergence insufficiency (esophoria) is best treated with prisms whose base is turned outwards; exercises are useless.

Couching.

G. LINDSAY JOHNSON (*Archives of Ophthalmology*, September, 1925) claims that the operation of couching a cataractous lens is sometimes justifiable. He admits that the sight obtained will last only a year or two, but he advocates it in old people who have lost one eye from chorioidal hæmorrhage following cataract extraction. His experience teaches him that when there are signs of diseased blood vessels, high arterial tension or general breakup of the system, the surgeon should hesitate in opening the eye and should seriously consider the advantages of couching.

Disturbances of Vision Due to Digitalis.

H. B. SPRAGUE, P. D. WHITE and J. F. KELLOGG (*The Journal of the American Medical Association*, September 5, 1925) report seven cases in which visual disturbance arose from excessive doses of digitalis. This condition was described as far back as 1785 by William Withering, who was the first to use the drug in effective doses. The earliest symptom is a flickering before the eyes, then the patient has greenish or yellowish vision and failure of sight. This is associated with nausea and vomiting and extreme muscular weakness.

LARYNGOLOGY AND OTOLOGY.

"Mercurochrome" in Otitic Diseases.

CHARLES TERRELL PORTER (*Boston Medical and Surgical Journal*, June 25, 1925) reports the successful employment of "Mercurochrome" by intravenous injection in cases of general septicæmia following mastoiditis and its complications. He estimated the full dosage as about five milligrammes per kilogram of body weight. He increased or decreased the amount and repeated the dose according to the initial reactions which showed wide variations. In one of his patients, aged twenty-five, a perisinus abscess was found during the performance of a simple mastoid operation, the sinus wall was thickened and the sinus was thrombosed and contained pus. Culture showed the presence of pneumococci. Two doses of "Mercurochrome" were given with three days' interval. Although the patient's con-

dition had been bad he made an excellent recovery. In the next patient, aged four years, the mastoiditis on one side was complicated by a peritonsillar abscess with granulations on the sinus and adjacent *dura mater*. On the other side the mastoid had broken down and was filled with pus and granulation tissue. The mastoids were operated on with three weeks' interval. A fortnight after the first operation "Mercurochrome" was given, also on the first and third day following the second operation. The patient made a good recovery. The third patient, a child of nine, with chronic suppurative *otitis media* had a simple mastoid operation performed on each side. For some time the patient had a septic fever and blood culture showed short chain streptococci. Septic arthritis of the right knee developed, but all symptoms cleared up under the intravenous exhibition of "Mercurochrome."

Chronic Otorrhœa.

ARTHUR J. WAGERS (*Atlantic Medical Journal*, September, 1925) lays stress on the necessity of cleansing thoroughly the external auditory canal and middle ear of pus, cerumen, cholesteatomatous masses, crusts *et cetera*, before making any curative application. He suggests wiping with a dry wool swab, then applying suction of 2.25 to 4.5 kilograms (five to ten pounds') pressure, again swabbing with a solution of alcohol and boric acid and wiping dry. Granulation tissue must be removed. Then germicides are applied. Colloidal silver preparations are satisfactory. Application of ultraviolet rays with the Kromayer lamp is often beneficial. Zinc ionization has not been very successful in his hands. "Mercurochrome 220 Soluble," proved successful in over 50% of the cases in which it was tried. He thinks the particular germicide used makes little difference in the final results obtained, the essential curative factor being thoroughness and frequency of treatment.

Chronic Progressive Deafness.

GEORGE C. CATHCART (*The Lancet*, May 9, 1925) reports the results of the use of the electrophonoide of Zünd-Burquet in the treatment of a hundred cases of chronic progressive deafness. The instrument reproduces the sound vibrations of the whole gamut of the human voice and thus gives a physiological stimulus to the ear. The sounds produced are of varying quality and are variable at will. The usual course of treatment consists of thirty sittings. Cathcart, however, gives twelve preliminary treatments. He claims that his results show that this method of treatment affords the most substantial advance in the treatment of chronic progressive deafness of recent years. He says 68% of his patients were definitely improved. These comprised 81% of those with nerve deafness, 67% of those with chronic *otitis media* and 55% of those with otosclerosis. All of these patients had been previously treated by ordinary methods and pronounced incapable of improvement. The records show that tinnitus is not only alleviated, but often dispelled.

Surgical Diathermy for Epistaxis.

C. HIRSCH (*Klinische Wochenschrift*, May 28, 1925) advises the use of surgical diathermy in cases of severe nasal hæmorrhage associated with arteriosclerosis. He gives the details of five cases in which all the usual methods of treatment including cauterization had failed. The average current used was three hundred and fifty to four hundred milliampères, though in one instance a current of five hundred milliampères was required.

Accessory Nasal Sinus Disease in Children.

CHARLES A. MCWILLIAMS (*New Orleans Medical and Surgical Journal*, August, 1925) states that during severe coryza, measles, scarlet fever, influenza and some of the other infectious diseases the sinuses of children are more or less involved, necessitating drainage. Sneezing is the most frequent symptom, particularly in the morning, next comes a dry unproductive cough, common at night, resembling whooping cough. Hoarseness and enlargement of the posterior group of cervical glands may occur. There is always a chronic post-nasal discharge. A thick muco-purulent discharge coming from the post-nasal space with an enlarged lateral band of lymphoid tissue is good evidence of sinus affection. The nasal mucosa is thick and boggy. The simple acute infections respond well to treatment—a laxative, rest in bed and nasal irrigation usually suffice, a water suction apparatus may prove useful. If diseased tonsils and adenoids exist, they should be removed and the antra irrigated.

Intracranial Complications of Suppurative Otitis Media.

C. F. YERGER (*Illinois Medical Journal*, October, 1924) states that of the total number of cases of suppurative *otitis media* occurring in the decade 1911-1920 at the Cook County Hospital, 10% had associated some form of intracranial complication. Multiple complications usually attended cases classified either as meningitis, sinus thrombosis or brain abscess. This multiple condition produces a composite type of clinical picture rendering diagnosis very difficult or impossible. Meningitis was found the most frequent complication—occurring in 5% of the cases. Sixty-three patients with meningitis showed a mortality of 97% and of the twenty-nine cases examined at autopsy 100% manifested meningitis, 34% sinus thrombosis, 14% cerebral abscess, 14% cerebellar abscess and 10% extra dural abscess. In both serous and purulent meningitis evidence of the pressure of an inflammation of the meninges is found in the spinal fluid, namely increased pressure, quantitative increase and qualitative change in the cytologic content of the fluid and the presence of globulin. A sterile fluid indicates a serous meningitis, but in the purulent type bacteria occur and in this type the fluid is characterized by a relatively high cell count, for example over 10,000. In serous meningitis the meningitic symptoms soon disappear after adequate drainage of the suppuration adjacent to the meninges. If they do not, some intra-cranial suppuration, either subdural, cerebral or cerebellar, may be suspected which calls for an immediate operative exploration of the middle and posterior cranial fossæ. If the meningococcus is found in the fluid an intra-spinal injection of antimeningococcic serum should be given. In sigmoid sinus thrombosis leucocytosis is usually pronounced and the fever is generally intermittent. A very suggestive sign in cases of sigmoid sinus and jugular bulb thrombosis is the presence of enlarged and tender submaxillary lymphatic glands. Brain abscess was found by Yerger in 2% of his cases and the symptoms were those of increased intracranial pressure and focal findings corresponding to the location of the abscess. The focal signs are those of disturbances of equilibrium, orientation and synergy. The most important procedure in connexion with the diagnosis of a suspected otitic intracranial complication is the examination of the cerebro-spinal fluid. Brain abscess should be operated on as soon as the diagnosis is made and it is best attacked *viâ* the avenue of the primary infection. Yerger recommends Ballance's two stage operation. Extradural abscess is rarely diagnosed, it is often associated with sinus phlebitis or thrombosis or both. Labyrinthitis occurred in less than 1% of the patients with suppurative *otitis media*.

Treatment of Non-Suppurative Deafness.

JAMES A. BABBITT (*Atlantic Medical Journal*, September, 1925) holds that fundamentally basic to all treatment of chronic non-suppurative deafness is the question of perception *versus* conduction priority. If the former has priority the toxæmic involvement of the auditory nerve and end organs means focal infection somewhere and unlimited investigation of septic foci is indicated. He considers that hyperplasia or exudative change is often overlooked. He quotes Holms to the effect that 90% of all the diseases of the middle ear are due to disease primarily in and about the tube. The fossa of Rosenmüller may dam up secretions by the contraction of the pharyngeal muscle. The relief of tinnitus is of importance in the treatment of deafness. He regards treatment of the Eustachian tube as the most promising type of local treatment. He concludes that the sum total of probable relief seems greater in conservation of hearing than in treatment of established deafness.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE WESTERN AUSTRALIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Perth Public Hospital on August 19, 1925, DR. T. L. ANDERSON, O.B.E., in the absence of the President, in the chair.

Radiology and the Gall Bladder.

DR. A. SYME JOHNSON read a paper entitled: "X Rays as an Aid in the Diagnosis of the Pathological Gall Bladder" (see page 545).

DR. DONALD SMITH said that he desired to express his appreciation of Dr. Johnson's able paper. He agreed that it was difficult to trace results. A radiographer sometimes heard when he was wrong but seldom when he was right.

In connexion with gall bladder radiography he would like to point out the value of the Potter Bucky diaphragm which consisted of a grid of plates moving across the film in such a way that they registered no image on it, while only the rays reaching the film in a radial line from their point of origin were allowed to pass through the diaphragm.

This was particularly useful in increasing shadow definition in stout people who were the type in which gall stones were most often found.

DR. SMITH showed three interesting films—one with large gall stones very clearly defined, the film having been taken with a Potter Bucky Diaphragm.

The second film corresponded to the first one and showed the same gall stone lying in relation to a semilunar indentation of the barium-filled duodenum.

The third film showed periduodenal adhesions and two small stones lying at a somewhat lower level than the pylorus.

The varying results which had been given in former years, were due to an excessive amount of reliance on direct examination of the gall bladder, for stone shadows which were often ill defined, while the indirect examination and signs were in his opinion much more valuable.

A collection of fæces sometimes gave shadows resembling stone shadows of this indefinite character and only shadows having well defined outline and the typical shape and arrangement of gall stones were to be considered of diagnostic value as direct signs.

The preparation of the patient was a difficult matter, for without preparation there were fæces to contend with and after enemata the colon was usually filled with gas.

He had found that it was best to feed the patient on light diet for forty-eight hours prior to examination and the aperient should be given forty-eight hours before and not repeated. He did not place as much reliance on colonic signs as he did on the appearance of the duodenal cap.

MR. F. A. HADLEY congratulated Dr. Johnson on what he termed the reasonableness of his views on the subject. Mr. Hadley thought that in discussions on the gall bladder more consideration should be given to pathology of the gall bladder itself than of gall stones. Gall stones were formed in groups, the relative ages of which could be told by their composition.

The early gall stones were of cholesterin, but later had a deposit of calcium on their surface. Thus whilst the older groups would show up, the earlier ones might not.

Mr. Hadley emphasized a danger in focussing the attention on gall stone shadows. If the shadows were not present on the film, the patient might refuse operation even when the indirect signs which were more reliable, gave definite proof of pathological changes in the gall bladder.

Another danger was that clinicians were liable to rely to too great an extent on radiography and to expect the radiologist to give a definite diagnosis. There was a danger of loss of clinical acumen. One test which had been claiming a lot of attention lately was that for the estimation of the cholesterol percentage in the blood a percentage of 0.125 to 0.150 was regarded as normal whilst anything above 0.2 was regarded as evidence of gall bladder infection. He called the attention of members to the excellent paper published in a recent issue of *The British Medical Journal* by Sir Berkley Moynihan on this subject.

DR. H. J. GRAY asked Dr. Johnson if pneumoperitoneum gave any assistance in the diagnosis of the pathological gall bladder.

Dextrocardia.

DR. P. WHITE showed a patient suffering from dextrocardia, who had come under his notice in the course of routine examination at the Repatriation Department. Clinically all the viscera were transposed, but no radiological examinations had been made.

Several members expressed surprise that such a condition should have been found in a returned soldier, but Mr. Hadley and Dr. Couch stated that several such individuals had been passed for active service and in the absence of any disability they saw no reason for rejecting such an applicant.

Pathological Specimens.

DR. W. J. BEVERIDGE presented pieces of skull removed twenty-five years previously from a South American Indian showing impacted hair in the fissured fractures. Dr. Beveridge exhibited these specimens as evidence of the elasticity of the human skeletal structure.

Infectious Mononucleosis.

DR. J. G. HISLOP read a paper entitled "Infectious Mononucleosis" (see page 557).

Gumma of the Tongue.

DR. J. J. HOLLAND showed a male patient, aged thirty-five years, who was suffering from gumma of the tongue, which had been present for three months. The ulcer had commenced to form at the posterior aspect and had moved towards the anterior part leaving a slight scarring in the track of the healing process. The differential diagnosis lay between malignant disease, syphilis, tuberculosis and actinomycosis. There were no glands palpable and whilst the patient admitted having had gonorrhœa, he had no knowledge of having had syphilis. However the blood gave a strong reaction to the Wassermann test and the patient was commencing treatment.

DR. J. K. COUCH said that this was probably a case in which primary urethral chancre had been mistaken for gonorrhœa. Patients affected by this type of chancre usually had a slight discharge which persisted for some weeks and could be diagnosed only on the passage of a sound.

A MEETING OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Alfred Hospital, Melbourne, on August 19, 1925, when a series of clinical demonstrations were conducted by the members of the Honorary Staff of the hospital.

Congenital Cyst of Liver.

MR. R. C. BROWN detailed the case history of a female patient, aged forty years, who was first admitted to the Alfred Hospital on September 6, 1922. She had then stated that she had been troubled with pain in the upper portion of the abdomen for a period of about six years. During the weeks preceding her admission to hospital the attacks had become more severe and of weekly frequency, some of them being distinguished by vomiting and jaundice. She complained of loss of weight and strength.

Six years previously this patient had been operated upon by Mr. T. P. Dunhill who told her that she "had a cyst which was very like hydatid but was of different nature."

On physical examination distension of the upper portion of the abdomen had been noticeable; it was apparently dependent upon the presence of a tumour the lower border

of which could be defined five centimetres below the umbilicus.

Mr. Brown had performed laparotomy on September 12, 1922. The liver had been occupied by a very large cyst from which 4.8 litres (four quarts) of thick grumous material were evacuated. The walls af the cyst had been trabeculated in parts and otherwise covered with soft, brown, papillomatous masses which bled freely on the slightest touch. Very numerous small grape-like pellucid cysts had been attached to the main cyst wall. Fifteen cubic centimetres of a 10% solution of formaldehyde had been introduced into the cavity of the cyst and the wall sutured.

A pathological report received from Dr. A. Trinca was to the effect that the cyst was a multilocular congenital cyst which had probably originated from maldevelopment of the bile canals.

The patient had been readmitted to hospital on December 17, 1922. She had complained of very severe pain and it was evident that she had lost much weight and strength. She had exhibited continued high fever and night sweats and a recurrence of swelling in the abdomen. The cyst had been opened again by operation and a drainage tube inserted but the patient had not immediately improved. A larger tube had then been passed through the cyst in such a manner that the ends came to the surface one on either side of the abdomen just lateral to the border of the *rectus abdominis* muscle. After this procedure the patient had made rapid improvement, but the tube was not removed until June 2, 1924. She had been in normal health since and was now apparently cured of the invalidism connected with the presence of the cyst.

Traumatic Osteitis of Lumbar Vertebra.

Mr. Brown's second patient was a woman, aged twenty-seven years, whose disability dated from a severe jolting which she sustained while crossing a double culvert in a motor car in March 1925. She had been seated in the rear seat of the car. Although shaken and in pain she had continued her journey for one hundred miles and at the end had descended from the car and walked inside unaided. On the following day she had been able to stoop and go about her duties as usual, but suffered backache and foot soreness. Both of these symptoms had gradually become aggravated and at the time of demonstration the patient could not stoop without experiencing severe pain in the back. She was unable to walk without assistance.

By physical examination great tenderness was elicited in the lumbo-sacral region; the tenderness was especially pronounced over the lower lumbar vertebræ and over the glutei muscles of the left side. No abnormal features were to be detected in the superficial and deep reflexes of the abdomen and lower limbs. The radiographer's report was to the effect that the film revealed destruction of the lower portion of the body of the fourth lumbar vertebra; also a calcified spur passing from the lower right hand corner of the body of the fourth toward the right border of the fifth lumbar vertebra.

Excision of Urethral Stricture.

Mr. Brown further discussed the clinical history of a male patient, aged fifty years. In 1919 this patient's left testicle had been removed on account of extensive tuberculous destruction which occasioned multiple sinuses. On August 24, 1924, he had been admitted to the Alfred Hospital for relief from retention of urine which occurred as the result of the presence of a urethral stricture of old standing. Two days later the stricture, together with two centimetres of urethra and much surrounding cicatricial tissue had been excised. A note recorded on October 27, 1924, stated that urination was free and that a large sound had been passed without any difficulty occasioned by urethral obstruction. On November 18, 1924, a discharge had appeared at the urethral meatus; microscopical examination of the material had shown that it contained very numerous pus cells but apparently no bacteria. By January 15, 1925, the operation wound had "broken down."

On August 5, 1925, this patient had again been admitted to hospital. He had displayed perineal and scrotal fistulæ with pus discharging from the meatus of the urethra and the scrotal opening.

Operation had been undertaken on August 13, 1925, when the fistulæ were laid open and examined. The scrotal sinus was found to communicate with the penile portion of the urethra and that in the perineum with the posterior portion of the urethra. A No. 14 sound had been passed through the urethra without difficulty.

Epithelioma of the Tonsil.

Mr. Brown's fourth patient was an elderly man who at the time of his admission to the Alfred Hospital in June, 1925, stated that he had suffered with sore throat for nine weeks and had noticed a lump in the left side of the neck for three weeks. For about the same period there had been a discharge from the left ear. Clinical examination showed the presence of a large epithelioma of the left tonsil, the growth spreading on to the fauces, alveolar ridge and adjacent cheek. Enlarged lymphatic glands had been apparent below the angle of the jaw on the left side.

Diathermy had been applied to the growth on the tonsil on June 23, 1925. On August 7, 1925, the cervical glands of the left side had been extirpated by Maitland's block dissection.

It was proposed to employ radium for the tonsillar growth which was showing indications of recurrence.

Mr. Brown also demonstrated another patient affected by epithelioma of the tonsil.

Urethral Stricture.

Mr. Balcombe Quick showed several patients in whom he had dealt with impassable stricture of the urethra by excision after the method of Mr. Hamilton Russell. The periods that had elapsed since the operations varied from two to eighteen months. In the case of the man who had been operated upon eighteen months previously no instrument had been passed during the last thirteen months and it was found that nothing larger than a Lister sound, No. 4-7, could be passed. Sounds varying in size from No. 10 to No. 13 could be passed without difficulty in all the other patients.

Ununited Fracture.

Mr. Quick also demonstrated from a child in whom much deformity of the leg had been occasioned by an ununited fracture of the tibia of nine years' duration. An operation for the insertion of a sliding graft had been followed by union, which however was temporary only. At a subsequent operation a massive graft from the opposite tibia had been fixed to the bone by bolting with Lane's plates and strong union had resulted. A series of radiograms was exhibited.

Supracondylar Amputation

Mr. Quick also showed a patient for whom he had carried out amputation of the thigh after the method of T. G. Orr. In this operation a flap of deep fascia from the posterior aspect of the thigh was brought to cover the end of the femur, thereby obviating adhesion of the deep surface of the anterior flap to the sawn end of the bone and ensuring a mobile covering.

Excision of the Tongue.

Mr. Quick's fourth patient was a man who had undergone complete excision of the tongue for carcinoma. The excision had been carried out at the level of the hyoid bone by means of the diathermy knife.

Decompression Apparatus.

Mr. Quick demonstrated the manner of use of the apparatus of Clay Shaw and H. H. Young for gradual decompression in the chronic vesical distension consequent on prostatic obstruction.

Ununited Fracture of Humerus, Paralysis of Radial Nerve.

Mr. Fay Maclure, O.B.E., showed a male patient, aged twenty-nine years, who sustained a fracture of the middle of the shaft of the humerus as a result of direct violence

on March 3, 1924. The radial nerve had been injured. Mr. Maclure had first seen the patient on August 5, 1924. No union had been present, the muscles had been wasted and massage had been given for six weeks. On September 16, 1924, a bone graft had been applied to the humerus. A graft, ten centimetres (four inches) long had been taken from the tibia. The radial nerve had been left alone in hope of restoration of function. By December 20, 1924, firm union of the humerus had occurred. Complete paralysis of the radial nerve had still been present. The patient had been told to use the arm and wear a "cock up" splint at night. When seen again on July 6, 1925, the radial nerve paralysis had still been complete. On July 8, 1925, operation had been undertaken. The ends of the radial nerve had been found separated by a gap of nearly 7.5 centimetres (three inches). An "amputation neuroma" had been found on the proximal end of the nerve and it had been impossible to bring the ends of the nerve together. Tendon transplantation had been performed. The *palmaris longus* tendon had been transplanted into that of the *extensor pollicis longus* and the tendon of the *flexor carpi radialis* into that of the *extensor communis*. The patient was having massage and reeducation movements.

Carcinoma of the Rectum.

Mr. Maclure's second patient was a man, aged thirty-six years, who had been admitted to hospital on October 13, 1924, suffering from carcinoma of the rectum. The patient had complained of diarrhœa, incontinence and unsatisfied defæcation for eleven weeks, together with the passage of bright red blood. On rectal examination multiple, firm, fairly mobile nodules had been discovered and an annular stricture above the nodules.

On October 18, 1924, a portion of the growth had been excised and a pathological report of carcinoma had been received.

On October 22, 1924, a laparotomy had been undertaken. Only the terminal portion of the rectum had been found involved. The rectum had been freely movable. No enlarged glands had been found on palpation. The wound had been closed.

On November 1, 1924, excision of the rectum had been undertaken. The anus had been closed. The incision had extended from the coccyx round the anus. The coccyx had been excised. The *levator ani* and the sphincter had been sutured to the gut and the peritoneum closed. Incontinence had been present for three weeks and then some measure of control had been obtained.

On December 30, 1924, the patient had been discharged from hospital with practically complete control. He had been readmitted for a patulous anus. After the patient had been standing for some time the gut had protruded for two and a half centimetres all round.

On July 8, 1925, diathermy had been used, incontinence had followed for two weeks and then complete control had been obtained. At the time of demonstration only one small portion was protruding.

Contraction of the Hand Following a Burn.

Mr. Maclure's third patient was a nurse, aged twenty-seven years, who suffered from contraction of the right hand. The hand had been crushed in a hot mangle on September 19, 1924. The palm had become lacerated. The wound had healed and had then started to contract. Mr. Maclure had first seen the patient on February 19, 1925. The condition of the hand at that time was demonstrated by a plaster cast. On February 21, 1925, a tube had been formed from the thigh. On March 20, 1925, a pedicle graft from the tube had been sutured to the palm after free excision of the scar tissue. The hand had been fastened to the side for one month and then the pedicle had been divided. The hand had been massaged and function restored.

On July 29, 1925, the patient had been seen again, when a small band of scar tissue had caused flexion contraction of the little finger. On July 30, 1925, the scar had been excised from the little finger and a pedicle graft from the tube on the thigh had been sutured in position. The hand had been fastened to the side. On August 15, 1925, the pedicle had been cut off from the tube and strapped to the little finger.

Crushed and Burned Hand.

Mr. Maclure showed in the fourth instance a patient, aged seventeen years, whose left hand had been crushed in a hot mangle. When first examined on June 10, 1924, the hand exhibited extreme scarring and contracture of the tissues of the dorsum and ankylosis of the metacarpo-phalangeal articulations.

The operative procedure adopted consisted of osteotomy of the metacarpo-phalangeal joints of the index and middle fingers and setting of these fingers at right angles to the palm. Scar tissue had been freely excised from the dorsum of the hand. The bones had been removed from the ring and little fingers and the soft tissues of these two fingers had been swung on to the dorsum and sutured to the bare areas.

A photograph and cast of the hand prior to operation were exhibited.

Diathermy.

Mr. Hugh Trumble, M.C., showed five patients to illustrate results obtained in the treatment of cancer by surgical diathermy.

In commenting upon the utility of diathermy Mr. Trumble said that its performance was easy, that the area to be treated was sterilized by the heat of the process with a consequent minimum of after infection; that healing was therefore more rapid and that diathermy as compared with other operative procedures was conservative of tissue. Inoperable growths might be attacked with very successful results, at any rate as far as the patient's comfort was concerned. Freedom from after pain was also a feature of treatment by diathermy.

Mr. Trumble's first patient was a man, aged forty-eight years, in whom a rodent ulcer had appeared on the ear in 1918. Diathermy had been applied on July 13, 1925. Photographs of the ulcer prior to treatment as well as two taken on July 20 and 30, 1925, subsequent to treatment were exhibited. Skin grafting had been carried out on August 3, 1925. The ulcer appeared clean and in a healing condition, being nearly covered by young skin. There was a sequestrum yet to separate and Mr. Trumble considered that further treatment would probably be necessary.

A similar stage had been reached in the case of a man, aged seventy-five years, who had been treated by diathermy for an epithelioma of the ear on July 13, 1925.

The effect of diathermy in the treatment of carcinoma of the lip and cheek was seen in another patient. An ulcer on the lower lip had made rapid growth during six months and had spread to the adjacent cheek; the ulcer was very foul, fungating in appearance and had occasioned the patient very severe pain. On May 28, 1925, the ulcer had been excised by the diathermy knife. From that date the patient had experienced complete relief from pain and healing had advanced rapidly. The area was now firmly healed and there was now no evidence of further trouble. A pathological report had confirmed the diagnosis of squamous celled carcinoma.

Carcinomata of the floor of the mouth and of the tongue had been treated in a man, aged sixty-eight years. In April, 1924, diathermy had been applied to a papillomatous growth in the floor of the mouth; local anæsthesia had been employed. A pathological examination had established the lesion as one of squamous celled carcinoma. In February, 1925, an ulcer had appeared on the margin of the tongue well away from the previous lesion. This also had been treated by diathermy under local anæsthesia and was found microscopically to be a squamous celled carcinoma.

The scars were scarcely visible and there was no sign of further trouble. No enlargement of the cervical glands could be detected and the patient was in excellent health.

Mr. Trumble also demonstrated from a man, aged sixty years, in whom a rodent ulcer of the face first appeared fifteen years previously. In spite of treatment by radium

and X rays at intervals the ulcer had advanced. Four applications of diathermy had been given between October 28, 1924 and June 19, 1925. At the commencement of treatment the ulcer had been foul and offensive; it had healed over the greater part of the surface. The malignant process had effected complete destruction of the upper lip and alveolus and a large part of both maxillary bones including the hard palate was destroyed. The interior of the maxillary antra could be seen on looking into the patient's mouth.

Fractures in Children.

Mr. H. Cecil Colville showed a series of patients in whom were illustrated the final results of the treatment of fractures of the region of the elbow joint in children.

Three of the patients had sustained fractures involving the lateral half of the lower epiphysis of the humerus and occasioning gross displacement of the capitellum. All three had been treated by open operation in which the procedure had been to replace the detached fragment and secure it in position by means of a single silver wire suture. One of these children had developed a radial paresis with a wrist drop a few days after operation, but this had completely disappeared after several weeks. During this period the hand had been kept on a "cock up" splint. In the same patient it had been necessary to remove the silver wire three months after operation as it was projecting through the skin. Each of the three children displayed an excellent anatomical and functional result.

In three other children supracondylar fractures of the lower end of the humerus had occasioned much backward and lateral displacement of the lower fragment and elbow joint. In these the treatment adopted had consisted in reduction under general anæsthesia, followed by maintenance of the arm in a positiion of full flexion at the elbow joint for three to four weeks. After the lapse of this time the arm had been kept in a sling for two weeks, the position being that represented by flexion of the elbow joint to a right angle. Full use of the arm had then been allowed. In each child active movements had been permitted from the beginning of treatment. In the later stages the return of extension had been assisted by requiring the patient to carry weights for a certain period each day. The results of treatment in this type of fracture were all good from a functional point of view with respect to the movements at the elbow joint, although anatomically they were not consistently perfect, as was shown by some bony irregularity of the lower end of the humerus.

In another patient the injury had been an oblique fracture of the middle portion of the shaft of the ulna, accompanied by dislocation of the head of the radius in a forward and lateral direction. The dislocation had been easily reduced with the aid of anæsthesia but had recurred very readily if the elbow joint was not maintained in the position of full flexion. Treatment had accordingly been carried out in this position without any splint or other apparatus having been applied to the ulnar fracture. In the final result the ulnar fragments were firmly united and the head of the radius was in good position.

Dr. Colville indicated that in none of these patients had massage or passive movement been allowed at any stage of the treatment.

Macrodactyly.

Mr. R. St. Clair Steuart showed a patient, thirteen years of age, who was suffering from macrodactyly. At birth the patient had apparently been normal, but at the age of six months an excessive growth of the thumb and index and middle fingers of the right hand had been noticed. At the age of twelve months overgrowth and deformity of the toes had commenced. Mr. St. Clair Steuart pointed out that the patient suffered from kyphosis and scoliosis. The thyreoid gland was enlarged. A firm, immovable and painless mass was present in the left side of the neck. The thumb and index and middle fingers of the right hand were greatly enlarged and deformed. The left patella was enlarged to a considerable extent. The feet were overgrown and deformed, the left more than the right. On X ray examination of the hands, feet and limbs extraordinary osteoarthritic changes in all the joints were found. The anteroposterior and vertical diameters of the *sella turcica* were roughly ten millimetres. The clinoid processes were plump and rounded, but seemed normal. The floor of the *sella turcica* was smooth and no double contour could be seen. The *sella turcica* was probably normal.

Aortic Lesion.

Dr. A. V. M. Anderson discussed the clinical features presented by a man, aged fifty-seven years. He had formerly been a labourer but had been in receipt of the invalid pension for eleven years. He had been admitted to the Alfred Hospital on July 26, 1925, as he displayed indications of cardiac failure.

By inquiry it had been elicited that the patient had contracted gonorrhœal infection and a penile sore of doubtful nature thirty-three years previously. Six years later he was said to have had rheumatic fever. His wife had borne two children each of whom had lived a few days only. He had been unable to work for eleven years and during that time had suffered from shortness of breath and had been affected intermittently by œdema of the ankles. He complained that recently he had been overcome by feelings of suffocation which were accompanied by præcordial pain.

In the clinical examination it was noted that the patient's right pupil was larger than the left, the latter reacting poorly to light and accommodation. The cardiac apex beat was located at a point 16.5 centimetres from the mid-line of the sternum. Systolic and diastolic murmurs were audible over the præcordium, but were heard best in the aortic area. The fingers were clubbed and a considerable degree of anæmia was apparent. Normal responses were obtained on testing the various deep tendon reflexes. It had been determined that the urine contained many leucocytes, but there appeared to be no other pathological constituents. Dr. J. Ringland Anderson had made an ophthalmological examination and had reported that the left eye was affected by old iritic adhesions. No evidence of syphilis had been obtained when this patient's blood was subjected to the Wassermann test.

Dr. Anderson said that the doubtful factor in this patient was that of ætiology; it was not clear whether the cardiac condition was to be attributed to rheumatism or syphilis.

Stenosis of the Œsophagus.

Dr. Anderson's second patient was a man, aged sixty years, who had been admitted to the Alfred Hospital on June 22, 1925, with the provisional diagnosis of alcoholic cirrhosis of the liver and pneumonia. The patient confesssed to having been addicted to alcohol for years. He had been married thirty years and had five healthy children whose ages ranged from thirty to twenty-one years.

The patient gave the history that a fortnight before his admission to hospital he had while intoxicated taken liquid from a wrong bottle. Following this he had vomited much blood for several days and during this time he had suffered severely from pain in the abdomen and in the left side of the chest. He also stated that he expectorated much thick phlegm, was very short of breath and generally in a state of exhaustion.

Clinical examination at the time of his admission to hospital had revealed signs of widely diffused bronchopneumonia. *Pyorrhœa alveolaris* had been noted. The tendon reflexes at the knee joint could not be elicited; no tubercle bacilli had been found in the sputum. An estimation of the urea content of the blood gave the figure thirty-five milligrammes per hundred cubic centimetres and by the urea concentration test he had been found to eliminate 3% of urea in the urine passed during the second hour after the ingestion of urea. The patient's clinical condition had gradually improved, the physical signs in the lungs clearing away and the tendon reflex at the knee returning to normal.

On July 12, 1925, he had complained of difficulty in swallowing and this had gradually progressed. He had been unable to take a barium meal. The blood had been sent for examination by the Wassermann test at this stage and a "positive" finding had been recorded.

Dr. Bryan Foster had made an examination of the patient under anæsthesia and had found definite œsophageal obstruction at the level of the second rib. The mucosa had been very much thickened and the appearances generally had been suggestive of malignant disease. Radiological examination had shown considerable widening of the aortic shadow to the right and left of the middle line in both horizontal and right oblique views.

Dr. Anderson discussed the condition of this patient with reference to simple, syphilitic and malignant stenosis of the œsophagus. Antisyphilitic treatment had been instituted.

Pernicious Anæmia.

Dr. Henry Laurie discussed the case record of a woman whom he had intended to show at the meeting. She was fifty-six years of age and had first come under his observation eight months previously. The salient features of her ill health at that time had been weakness and asthenia, diarrhœa and soreness of the mouth and tongue. She had been troubled with diarrhœa at intervals since then and from time to time had complained of indefinite tingling in the hands and feet. She presented a lemon yellow colouration of the skin, much emaciation and a reddish and glazed tongue. The spleen could not be shown to be enlarged.

Examination of the blood had revealed that the red blood cells numbered 820,000 per cubic millimetre,the hæmoglobin value registered 25%. The colour index was therefore 1.5. Features of the film were decided anisocytosis and poikilocytosis. There were many oval macrocytes, stippled red cells and others exhibiting diffuse polychromatophilia. Many nucleated red cells occurred.

Gastric analysis had disclosed complete achlorhydria. A positive finding had been recorded when the Fouchet test was applied to the blood serum.

This patient had died on August 7, 1925.

Pernicious Anæmia in Remission.

Dr. Laurie presented a woman, aged sixty-two years, who had been conspicuously pale for twelve months. One month previously she had first complained of weakness and a feeling of tightness in the chest. She had also described sensations of tingling in the left arm, the abnormal sensations extending to the fingers which had then become numb. Her vision was much impaired.

Clinical examination had shown that the visual disturbance was based on retinal and vitreous hæmorrhages. There was no indication of glossitis and no history of diarrhœa could be elicited. The tingling and numbness complained of were apparently subjective only as no neurological signs could be detected on examination.

When the blood was examined on June 26, 1925, it had been found that the red and white corpuscles numbered 1,200,000 and 6,400, per cubic millimetre respectively, the percentage of hæmoglobin was 35, the colour index being 1.4. By a differential count of the leucocytes the polymorphonuclear cells had been estimated as 23% of the total, the myelocytes as 8% and the remaining 69% had been comprised of cells of the lymphocytic series. The film had exhibited poikilocytosis and anisocytosis, a relative lymphocytosis and occasional nucleated red cells.

Examined again on July 30, 1925, the blood had shown an increase in the red cells to 2,500,000 per cubic millimetre. Hæmoglobin had advanced to 60% and there had been a fall in the colour index to 1.1.

Complete achlorhydria had been demonstrated by gastric analysis and the result of the Fouchet test, faintly positive on the occasion of the first examination of the blood, had been an absence of reaction on the date of the second examination.

Dr. Laurie regarded this patient on clinical grounds as affected with pernicious anæmia. She was improving under arsenical medication.

Duodenal Ulcer: Gastro-Enterostomy: Closure of Stoma.

Dr. Laurie's second patient was a man, aged forty-one years, who six years previously had undergone gastro-enterostomy for duodenal ulcer. Symptoms had recurred four years later and at this time melæna had been noted. X ray examination carried out then had shown that there was delay in the emptying of the stomach.

This patient had been admitted to the Alfred Hospital on June 3, 1925, having suffered from a severe hæmatemesis within the preceding twenty-four hours. By X ray examination a duodenal cap had been observed and it had been determined that the stoma of the gastro-enterostomy was not functioning.

One month after his admission to hospital the patient's red blood corpuscles had been found to number 3,200,000 per cubic millimetre. Hæmoglobin had been reduced to 60%, the colour index being 0.9. There was no record of gastric analysis. No response had been obtained to the Fouchet test applied to the blood serum. On July 14, 1925, Mr. Balcombe Quick had resected the closed stoma and had restored the condition of gastro-enterostomy.

Banti's Disease.

A male patient, aged twenty-seven years, was presented by Dr. Laurie as furnishing an example of Banti's disease. Twenty-four hours before admission to hospital on May 25, 1925, this man had vomited 600 cubic centimetres (one pint) of blood. It had been ascertained that for twelve months previously he had been troubled by epigastric pain at intervals. The pain had supervened at an interval after food varying from one-half to three hours and was especially likely to follow highly seasoned foods. Such pain had been definitely worse during the preceding three months. There had been no vomiting and no loss of weight or appetite. Physical examination had shown that the spleen was very much enlarged. Normal findings had attended an X ray examination of the gastro-intestinal tract.

Detailed examination of the patient's blood on various occasions had revealed anæmia of a non-distinctive type. The observations included an investigation of the fragility of the red corpuscles, but hæmolysis had not occurred beyond 0.44% saline solution. Gastric analysis had yielded no pathological findings and by the Fouchet test no bile pigment had been detected in the blood serum.

He had been discharged from hospital on June 12, 1925, to be admitted again on July 4, on account of recurrence of hæmatemesis. In the interval he had been comparatively well. The spleen was enlarged as before. No indication of syphilitic taint was afforded by the Wassermann test.

Lipiodol.

Dr. J. F. MacKeddie gave a detailed demonstration of the manner in which intrathecal injections of "Lipiodol" were employed in the localization of tumours and other lesions of the spinal cord and meninges. A number of patients in whom the method had been employed were shown as also the radiograms taken after the injection of "Lipiodol." Dr. MacKeddie discussed in detail the case histories and neurological signs in each of the patients.

Mitral Stenosis.

Dr. M. D. Silberberg showed a male patient, aged fifty years, who had suffered from postrheumatic auricular fibrillation in March, 1922. Normal rhythm had been restored at that time by the use of quinidine sulphate. The heart had remained normal until May, 1925, when fibrillation returned while the patient was dancing. Quinidine treatment had then converted the fibrillation into auricular flutter. A course of digitalis had then caused a return of fibrillation. A further course of quinidine had restored normal rhythm. Dr. Silberberg pointed out that the apex beat was in the sixth intercostal space and 12.5 centimetres (five inches) from the middle line. Mitral presystolic, systolic and diastolic bruits were present during normal rhythm.

Recurrent Ventricular Paroxysmal Tachycardia Associated with Syncope.

Dr. Silberberg also showed a male patient, aged twenty-five years, an iron moulder, who complained of "peculiar

turns" since December, 1924. The patient often experienced as many as eight "turns" in an hour. While in the "turns" he might lose control of his legs suddenly and had fallen and become unconscious, sometimes he had experienced a feeling of weakness and giddiness. The "turns" lasted merely a few seconds. He had noticed that the heart might drop beats or that its action might be rapid for short periods. The patient's general health was good and his habits were temperate. He had had two attacks of rheumatic fever at the ages of eight and twelve years. His serum had not reacted to the Wassermann test. The patient was a tall man of good colour and well nourished. The cardiac apex beat was 13.1 centimetres (five and a quarter inches) from the middle line. The sounds were clear. Frequent short periods of tachycardia for about four to six or more beats occurred, sometimes the abnormality was confined to the occurrence of extra systoles only. Electrocardiographic records showed a series of right ventricular extra systoles, in other words a short paroxysmal tachycardia attack from a focus in the right ventricle. During the paroxysm the unpleasant symptoms occurred, they depended on the duration of the attack.

Complete Heart Block.

Dr. Silberberg's third patient was a male, aged thirty-three years, who was suffering from complete heart block. His usual ventricular rate was forty-eight in the minute. He had suffered from a number of severe syncopal attacks (Stokes-Adams disease). Three years previously he had been treated for pericarditis and had afterwards been suspected of suffering from subacute infective endocarditis. For about eighteen months he had had a frequent evening rise of temperature to 37.2° C. or 37.8° C. (99° F. or 100° F.). He had rested in a wheel bed for nearly two years. At about this time syncopal attacks had begun to occur. The pulse rate had varied from forty-eight to sixty-six in the minute and electrocardiographic records had revealed complete heart block pressure. A harsh systolic bruit of maximal intensity in the apical region had become more noticeable in the preceding twelve months. The patient's general condition was good, he had had no recent syncope, his pulse rate was usually forty-eight in the minute and the electrocardiographic record revealed complete heart block.

Mitral Stenosis with Auricular Fibrillation

Dr. Silberberg also showed a male patient, aged forty years. The patient had suffered from auricular fibrillation and mitral stenosis more than two years previously. Normal rhythm had been successfully restored with quinidine sulphate. Over one year later the fibrillation had occurred after the patient walked up the Flinder's Street railway ramp. He had responded to five doses of quinidine. Four months later during a severe attack of bronchopneumonia fibrillation had again been present. During early convalescence while toxic symptoms were manifest quinidine had failed to correct the rhythm. A month later normal rhythm had again been restored. At the time of demonstration the rhythm was still normal. There were signs of mitral stenosis including a presystolic bruit, a slapping first sound and a diastolic bruit. There was a history of rheumatism in childhood.

Meningocele.

Dr. Walter Summons, O.B.E., showed a male patient, aged twenty-one years, who had been well until four years previously. At this time he had begun to complain of pain in the back of the thighs and the legs. He had suffered from incontinence of urine for eighteen months, defæcation had been normal, but the patient noticed wasting of the left leg during the previous nine months and had suffered from a spasm of the muscles of the back of the left thigh and calf. He complained of numbness of the left buttock and hypersensitiveness down the backs of both thighs and legs. He could not stoop without first bending the knees. On examination a spastic paresis of the hamstring and calf muscles was found. This was more prominent on the left side and was accompanied by wasting. The knee jerks were exaggerated and the *tendo Achillis* jerk was present. The anal reflex could not be obtained. A loss of sensation was present over the distribution of all the sacral segments. The bladder was empty and incontinent dribbling was present. A fluctuant tender swelling was present over the upper sacral vertebræ. A skiagram revealed the presence of a sacral *spina bifida*. The serum had not reacted to the Wassermann test. A diagnosis of meningocele of the lower end of the spinal membrane had been made.

Cortical Degeneration of Uncertain Origin.

Dr. Summons also showed a male patient, aged forty-five years, an engineer, who was married and had three healthy children. The patient had been ill for two years. The onset of illness had been gradual, but at intervals the patient had experienced a number of sudden "turns" followed by definite extension of symptoms. The patient was moody, emotional and suffered from loss of memory. The speech was slow, but no apparent aphasia was present. No paralysis was present. The control of the sphincter was erratic. On examination it was seen that the patient had a spastic gait. Ataxia was present and spasticity was general, but more pronounced on the right side. All the tendon reflexes were exaggerated, but more so on the right side. Ankle clonus could be elicited. The plantar reflex was of the extensor type on the right side and of the flexor type on the left. The superficial abdominal and cremasteric reflexes were present. The eyes reacted to light and accommodation. Lateral and rotatory nystagmus was present. No sensory loss either superficial or deep could be detected. The serum had not reacted to the Wassermann test. The systolic blood pressure was 190 and the diastolic 150 millimetres of mercury. The father had died of apoplexy and the mother of some slow nervous degeneration. Dr. Summons regarded the condition as a cortical degeneration of uncertain origin.

Amyloid Disease.

Dr. J. F. Chambers presented as affected with amyloid disease a man, aged thirty-six years. He was a Russian and a total abstainer from alcohol. He had had a small discharging sinus on the right side of the neck, the outcome of tuberculous disease of the cervical vertebræ for four years. Despite the presence of the sinus and the underlying disease the patient had always felt in good health and he remained free from symptoms except the slight discomfort occasioned by swelling of the abdomen which he had noticed for the previous twelve months.

On physical examination both liver and spleen were found much enlarged; the enlargement was uniform and was associated with firmness, but no tenderness of the organs. There had been no jaundice or ascites. Polyuria was a feature and the urine contained albumin. The patient's systolic and diastolic blood pressures were 140 and 100 millimetres of mercury respectively. There had been no gastrointestinal manifestations. No history could be obtained of venereal or tropical disease. On two recent occasions the patient's blood serum had been examined by the Wassermann test and no response had been obtained. Cytological examination of the blood and an investigation of the corpuscular fragility had disclosed no abnormal features.

Amyotonia Congenita (Thomsen's Disease).

Dr. F. Trinca showed a male patient in whom the features of Thomsen's disease were well illustrated. All his life this patient had been subject to distressing attacks which were provoked by sudden muscular activity. If he hurried an attack would be ushered in with twitching followed by paresis in the legs; he suffered from palpitation and dyspnœa and finally amaurosis and vertigo. After the enforced rest the symptoms passed off, but they were very prone to recur on slight exertion. He could induce rapid exhaustion of the muscles of his arms by a few minutes' exercise with a punching ball.

Of five members of his family one only, a brother, was healthy and free from amyotonia. One sister in particular was very badly affected to such a degree that paralysis of the muscles of mastication was apt to ensue when she was eating.

Goitre.

Dr. Trinca's second patient was a young woman who had exhibited swelling of the thyreoid gland for two years. With this were associated exopthalmos, tachycardia and præcordial pain. Since May 14, 1925, this patient had been treated by endocrine therapy. Her basal metabolic rate which had formerly been —4 had been converted to —8 and her pulse rate had been reduced from an average rate of 130 per minute to 78. There had been great reduction in the swelling of the thyreoid gland and in general a return to normal health. The treatment employed had consisted in the administration of one ovarian tablet daily for seven days and a half tablet of spleen extract daily for four days. These measures had been suspended during alternate weeks.

Rhachitic Sequelæ.

Dr. Trinca also showed a man as manifesting rhachitic sequelæ in middle life. These embraced: (i.) Various cardiac disabilities and the "effort syndrome," (ii.) rhachitic defect of chest and defective expansion, (iii.) a tendency to visceroptosis, (iv.) neurasthenia, (v.) hypothyreoidism.

Pemphigus.

Dr. S. W. Shields showed a woman, aged forty-three years, who furnished a good example of pemphigus. Bullæ had first developed ten days previously, appearing on the neck and scalp; they had since spread rapidly over the trunk to the thighs. The lesions which were generally about two centimetres in diameter, exhibited yellowish contents although some were hæmorrhagic; they appeared on apparently healthy skin. The patient felt ill and depressed, but was afebrile. The fluid from the blebs had exhibited no excess of eosinophile leucocytes.

Urticaria Pigmentosa.

The macular type of *urticaria pigmentosa* was well illustrated in a girl aged six years. Brownish macules were scattered profusely over the trunk and thighs. The first urticarial attack in this child had occurred when she was a few months old and although she had no attacks of irritation the lesions became urticarial on the application of friction.

A woman, aged forty years, who was suffering with the same condition had been unable to come to the meeting. In her case the lesions had first appeared two years previously.

Bazin's Disease.

Dr. Shields demonstrated the lesions of Bazin's disease in a female patient, aged twenty-one years. Tuberculides of the hypoderm, symmetrically affecting the calves and outer sides of the legs, were the diagnostic features. The patient had been afflicted with recurrent ulceration of these parts each winter for the preceding seven years, but this year was free from ulceration. The lesions commenced as purplish-red nodes many of which progressed to somewhat deep ulcers. These ulcers exhibited sharp irregular edges and some surrounding infiltration. This patient had tuberculous cervical glands and exhibited a "chilblain" circulation of the lower extremities. General hygienic measures and local treatment had been instituted in 1925 well in advance of the winter months with successful results.

Rodent Ulcer.

Rodent ulcer in a girl, sixteen years of age, was demonstrated by Dr. Shields. The ulcer, situated on the left cheek, was typical as regards the edge and general appearance. It had been first noticed by the patient's mother two years previously and had been increasing in size since then.

Lichen Planus Atrophicus.

Lichen planus atrophicus was exemplified in a woman, aged sixty years. Multiple atrophic patches of pearly whiteness were present on the skin over both wrists; in the case of some of the patches minute horny plugs were visible at the mouths of the follicles.

Ichthyosis.

A boy, aged four years, furnished an example of ichthyosis. The condition had first been noticed when he was two days old and the boy exhibited the characteristic generalized dryness of the skin and scattered areas on which were fish-like scales.

Adenoma Sebaceum.

Dr. Shields showed a female patient, aged nineteen years, as illustrating *adenoma sebaceum* (Pringle type). He demonstrated circumscribed overgrowth of sebaceous glands and vascular elements appearing as innumerable small reddish tumours which were best seen on the nasolabial furrows and on the chin. There was no history of fits, but the patient was mentally very dull and was becoming progressively more so. Renal function tests were attended by normal findings. No response had been obtained to the Wassermann test.

Darier's Disease.

Darier's disease was illustrated in the case of a female patient, aged forty-four years. The first skin lesions had appeared when the patient was sixteen years old and were of the character of follicular, crusted papules. Under the greyish-brown crusts were the dilated sebaceous orifices. The skin had a roughened and dirty appearance. The worst of the affection was to be seen on the chest, back, forearms and in the axillæ where the lesions gave rise to a very offensive odour in warm weather.

Molluscum Fibrosum.

A man, aged fifty years, furnished an example of *molluscum fibrosum*. Pigmentation and multiple fibrous tumours were to be observed in the skin of the face, trunk and limbs. The pigmentation had first been noticed soon after birth and the tumours three or four years later. Small lesions were palpable along the nerve sheaths. The patient's mentality was good.

Striæ Atrophicæ.

A man, aged thirty years, was shown in whom were seen soft and and indentable livid streaks which had appeared for the first time on both flanks two days previously. The lesions were of smooth surface and bluish red colour; their borders were very distinct and they were characterized by atrophy of elastic tissue. The man had gained 12.7 kilograms (two stone) in weight in the course of the previous few weeks.

Lesions of the Optic Nerve.

Dr. J. Ringland Anderson had assembled a number of patients for the purpose of demonstrating various lesions of the optic nerve which might be mistaken for optic atrophy. These included opaque nerve fibres, myopic crescents, congenital strands of connective tissue veiling portion of the disc. Fundi with wide but physiological cups were also exhibited.

An interesting patient was a woman who, although her discs were completely white, was possessed of normal vision and full fields. Six years previously she had had an illness which was probably disseminated sclerosis. When she came under observation at that time there had been a history of two weeks' duration of failing vision, headaches and weakness of the legs. Vision had been reduced to the perception of hand movements. The superficial abdominal reflexes had not been elicited, while all the deep tendon reflexes were exaggerated; the response to the plantar reflexes had been extensor in character on both sides. On withdrawal of cerebrospinal fluid by lumbar puncture it had been under normal tension. No information of a positive character had been yielded by the examination of the fluid, the investigation embracing the Wassermann test.

The patient had remained two months in hospital and on her discharge the vision in the right eye had been $^6/_{18}$ and in the left eye $^6/_{60}$. Three years later the vision had been normal in both eyes and at the time of the meeting vision in both eyes was represented by $^6/_6$ and the fields were full. Dr. Anderson suggested that the whiteness of the discs was due to the formation of neuroglia. The patient was healthy and normal in all respects.

Dr. Anderson also demonstrated optic atrophy due to diverse causes in patients affected by glaucoma, tabes, re-

tinal degeneration, otitic meningitis and tumour in the base of the skull.

Changes in the disc selected as likely to be mistaken for papillœdema were shown as they occurred in pseudo-neuritis, thrombosis of the central retinal vein and albuminuric retinitis.

One of the subjects of nephritis was a girl, fourteen years of age, whose early symptoms comprised headaches, vomiting and failing vision. Her systolic blood pressure was the equivalent of 190 millimetres of mercury. Albumin was present in the urine. Gross papillœdema was to be observed on ophthalmoscopic examination on both sides and the lower half of the retina in each eye was completely detached. Numerous hæmorrhages and areas of exudate were also present.

There were in addition patients whose fundi exhibited characteristic pictures of diabetic and arterio-sclerotic retinitis.

Disseminated retino-chorioiditis was demonstrated in two patients in whom no stigmata of syphilis could be detected by clinical examination or by the Wassermann test.

Radiography.

Dr. C. E. Dennis and Dr. L. Love demonstrated from a gallery of radiographic films effectively displayed in the X Ray Department.

Epithelioma of the Larynx.

Dr. H. Barry Thomson showed a man, aged sixty-five years, who was under treatment for epithelioma of the larynx.. The growth as it affected the larynx was of extrinsic origin, extending from the base of the left tonsil to the epiglottis and vocal cords. The patient was receiving the benefit of buried radium and deep X ray therapy.

Lesion of the Larynx.

Dr. Thomson's second patient was shown in order to obtain suggestions regarding diagnosis. He was a man who had been affected by hoarseness for the past eight years, but it had become much aggravated during the last six months. There was no ulceration of the larynx, but the whole of the left side was infiltrated, turgid and swollen. The blood serum had been examined by the Wassermann test, but no reaction had been obtained.

Lesion of the Nose.

A girl, aged nine years, presented a hard swelling on the right side of the nose; it occasioned her no pain, but was gradually increasing in size. A radiogram showed a slight increase in density in the ethmoid bone of the affected side. Both canine teeth in the upper jaw were unerupted.

NOMINATIONS AND ELECTIONS.

The undermentioned has been nominated for election as a member of the New South Wales Branch of the British Medical Association:

Janes, Arthur Fitzgerald, M.B., Ch.M., 1925 (Univ. Sydney), Wychwood, Turramurra.

Correspondence.

IS FEE SPLITTING COMMON IN AUSTRALIA?

Sir: For quite a long while I have intended to ask the Victorian Branch of the British Medical Association to investigate this important matter, but it is extremely difficult to get authentic cases to put before the Council.

These are just feathers of suspicion blown hither and thither by gusty unethical winds that make the observant consider. Perhaps these gusts are developing into a steady trade wind. For instance a few years ago a friend of mine was pilloried for having suggested to a surgeon that fees should be split. He was most apologetic and explained that all practitioners in his environment split fees and he thought it the accepted thing. Three years ago I had within six weeks six cases sent to me from the country by one practitioner. They were extraordinarily good gynæcological cases and all paid remunerative fees. Incidentally the postoperative results were excellent. Suddenly the flow of cases stopped. On making careful inquiries I had almost enough evidence to show that the country practitioner ceased to send me cases as he did not receive half my operative fees.

A specialist friend of mine stated a price to a general practitioner for an abdominal operation. The general practitioner collected the fee and sent the specialist half his fee reserving the rest for himself. A young man in practice with medical tendencies quite recently told me that affairs were going better with him as he had arranged with another young friend with surgical aspirations to split fees.

A few vague hints like this do make me consider if it is a normal condition here; certainly closely and carefully guarded. I have before me the *Bulletin* of The American College of Surgeons in which it says:

> It is particularly important that each fellow should have a thorough knowledge of the pledge of the College and particularly that part which has to do with commercialization of medical practice and division of fees.

The Pledge may be quoted as follows:

> I pledge myself as far as I am able, to avoid the sins of selfishness, to shun unwarranted publicity, dishonest money-seeking and commercialism as disgraceful to our profession; to refuse utterly all money trades with consultants, practitioners or others; to teach the patient his financial duty to the physician and to expect the practitioner to obtain his compensation directly from the patient, to make my fees commensurate with the service rendered and with the patient's rights and to avoid discrediting my associates by taking unwarranted compensation.

I think in conclusion it would be very interesting to open your columns for correspondence of this important subject.

Yours, etc.,

A. Norman McArthur.
M.B., B.S., M.R.C.S., L.R.C.P., F.A.C.S.,
Senior Gynæcological Surgeon to In-patients, Saint Vincent's Hospital, Melbourne.
74, Collins Street, Melbourne,
October 14, 1925.

THE JOHN IRVINE HUNTER MEMORIAL FUND.

The committee of the John Irvine Hunter Memorial Fund has circularized the members of the several Branches of the British Medical Association in Australia calling attention to the proposals that have been adopted for the perpetuation of the memory of the late John Irvine Hunter. These proposals were set out in The Medical Journal of Australia of September 12, 1825. Members are invited to contribute to the fund.

The list of subscriptions to October 31, 1925, is given below:

	£	s.	d.
R. Ross, Esq.	250	0	0
Messrs. Angus & Robertson	200	0	0
Dr. E. W. Fairfax (first contribution)	100	0	0
Dr. Keith Inglis	100	0	0
Dr. Gordon Craig	50	0	0
Dr. & Mrs. H. R. G. Poate (first contribution)	50	0	0
Dr. G. H. S. Lightoller	50	0	0
Professor A. E. Mills	50	0	0
Dr. N. D. Royle	50	0	0
Sir Alexander MacCormick	50	0	0
Dr. C. Bickerton Blackburn	50	0	0
Dr. Ralph Noble	25	0	0
Acting Professor Maguire	25	0	0
Dr. R. H. Todd	25	0	0
Dr. H. J. Nixon	25	0	0

	£	s.	d.
Professor A. N. Burkitt	20	0	0
Dr. B. Coen	20	0	0
J. Dunlop, Esq.	15	0	0
The Honourable Philip Whistler Street	10	10	0
Sir Henry Braddon	10	10	0
Judge Backhouse	10	10	0
W. P. Dunlop, Esq.	10	10	0
Professor MacCallum	10	10	0
Babcock & Wilcox, Limited	10	10	0
Mr. & Mrs. Leslie A. Denison	10	10	0
D. Mackay, Esq.	10	0	0
Lady Mackellar	5	5	0
Dr. F. Brown Craig	5	5	0
Professor E. Holme	5	5	0
Dr. O. Latham (first contribution)	5	0	0
Dr. R. B. P. Monson	3	3	0
Mr. L. Schaeffer	3	3	0
Miss E. M. Dibbs	3	3	0
Dr. W. M. A. Fletcher	3	3	0
Dr. V. Rich	3	3	0
Dr. W. Mawson	2	2	6
Dr. R. W. Heggaton	2	2	6
Dr. H. R. Hodgkinson	2	2	6
W. R. York, Esq.	2	2	0
A. Sutton, Esq.	2	2	0
Professor H. S. Carslaw	2	2	0
Miss M. A. Bailey	2	2	0
Miss L. Collins	2	2	0
H. A. Russell, Esq.	2	2	0
A. H. Lewes, Esq.	2	2	0
Dr. H. Daly	2	2	0
John A. Milford, Esq.	2	2	0
Dr. W. C. Kerr	2	2	0
Dr. A. L. North	2	2	0
Dr. F. L. Macqueen	2	2	0
Mrs. A. Constance Harwood	2	2	0
E. M. Wellish, Esq.	2	0	0
Dr. A. B. Nairn	2	0	0
G. A. Cantello, Esq.	1	1	6
L. G. H. Watson, Esq.	1	1	0
C. H. Goldie Simpson, Esq.	1	1	0
W. Cope, Esq.	1	1	0
Miss D. J. Smith	1	1	0
Miss M. Armstrong	1	1	0
The Rev. E. E. Anderson	1	1	0
Mrs. W. Renshaw	1	1	0
Miss F. M. Nowell	1	1	0
Mrs. A. Stewart (first contribution)	1	1	0
The Rev. Morris G. Fielding	1	1	0
H. T. E. Holt, Esq.	1	1	0
C. E. Martin, Esq.	1	1	0
J. H. Reid, Esq., B.Ec.	1	1	0
O'Donnell, Griffin & Co.	1	1	0
Dr. A. Bulteau	1	1	0
Miss M. G. E. Latreille	1	1	0
Dr. V. Cousins	1	1	0
T. B. Nicol, Esq.	1	1	0
Dr. A. Mark Stanton	1	1	0
Dr. K. S. Macarthur Brown	1	1	0
Miss M. Jopling	1	0	0
Miss L. Layh	1	0	0
B. C. Doig, Esq.	0	10	6
F. H. Henningham, Esq.	0	10	0
H. F. Wilson, Esq.	0	10	0
R. Barnes, Esq.	0	10	0
Miss D. V. Coutts	0	10	0
G. D. Osborne, Esq.	0	5	0
	£1,339	10	6

Medical Appointments.

Dr. James Garnet Sleeman (B.M.A.) has been appointed Superintendent of the Adelaide Hospital, Adelaide.

* * * *

Dr. Geoffrey Alleyne Cook (B.M.A.) and Dr. Benjamin George Johnston (B.M.A.) have been appointed Resident Medical Officers at the Adelaide Hospital, Adelaide.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, 429, Strand, London, W.C.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham Dispensary. Manchester United Oddfellows' Medical Institute, Elizabeth Street, Sydney. Marrickville United Friendly Societies' Dispensary. North Sydney United Friendly Societies. People's Prudential Benefit Society. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane United Friendly Society Institute. Stannary Hills Hospital.
SOUTH AUSTRALIAN: Honorary Secretary, 12, North Terrace, Adelaide.	Contract Practice Appointments at Renmark and Murat Bay. Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Diary for the Month.

Nov. 10.—Tasmanian Branch, B.M.A.: Branch.
Nov. 10.—New South Wales Branch, B.M.A.: Ethics Committee.
Nov. 11.—Victorian Branch, B.M.A.: Branch; last date of nominations for Council. Election of Scrutineers.
Nov. 11.—Victorian Branch, B.M.A.: Clinical Meeting Children's Hospital.
Nov. 12.—Victorian Branch, B.M.A.: Council.
Nov. 12.—South Australian Branch, B.M.A.: Council.
Nov. 12.—New South Wales Branch, B.M.A.: Clinical Meeting.
Nov. 13.—Queensland Branch, B.M.A. Council.
Nov. 13.—Western Australian Branch, B.M.A. Council.
Nov. 16.—New South Wales Branch, B.M.A.: Organization and Science Committee.
Nov. 17.—Tasmanian Branch, B.M.A. Council.
Nov. 17.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
Nov. 17.—Illawarra Suburbs Medical Association, New South Wales.
Nov. 18.—Tasmanian Branch, B.M.A.: Branch.
Nov. 24.—New South Wales Branch, B.M.A.: Medical Politics Committee.
Nov. 25.—Victorian Branch, B.M.A.: Council; Ballot paper issued.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, Sydney. (Telephones: MW 2651-2.)

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are £2 for Australia and £2 5s. abroad *per annum* payable in advance.